GASTROENTEROLOGY

ABSTRACTS AND CITATIONS

NATIONAL INSTITUTE OF ARTHRITIS, METABOLISM, AND DIGESTIVE DISEASES

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis, Metabolism and Digestive Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology have accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis, Metabolism, and Digestive Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

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The Secretary of the Department of Health, Education, and Welfare has determined that the publication of this periodical is necessary in the transaction of the public business required by law of this Department. Use of funds for printing this periodical has been approved by the Director of the Office of Management and Budget through April 30, 1981.

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LANGUAGE ABBREVIATIONS

Afrikaans	Afr.	Georgian	Geo.	Persian	Per.
Albanian	Alb.	German	Ger.	Polish	Pol.
Arabic	Ara.	Greek	Gre.	Portuguese	Por.
Armenian	Arm.	Hausa	Hau.	Pushto	Pus.
Azerbaijani	Aze.	Hebrew	Heb.	Rumanian	Rum.
Belorussian	Bel.	Hindi	Hin.	Russian	Rus.
Bengali	Ben.	Hungarian	Hun.	Serbian	Ser.
Bulgarian	Bul.	Icelandic	Ice.	Slovak	Slo.
Burmese	Bur.	Indonesian	Ind.	Slovene	Slv.
Chinese	Chi.	Interlingua	Int.	Spanish	Spa.
Croatian	Cro.	Italian	Ita.	Swahili	Swa.
Czechoslovakian	Cze.	Japanese	Jpn.	Swedish	Swe.
Danish	Dan.	Kirghiz	Kir.	Tagalog	Tag.
Dutch	Dut.	Korean	Kor.	Tamil	Tam.
English	Eng.	Latin	Lat.	Telugu	Tel.
Esperanto	Esp.	Latvian	Lav.	Thai	Tha.
Estonian	Est.	Lithuanian	Lit.	Turkish	Tur.
Finnish	Fin.	Marathi	Mar.	Ukranian	Ukr.
Flemish	Fle.	Masai	Mas.	Urdu	Urd.
French	Fre.	Multilingual	Mlt.	Vietnamese	Vie.
Gaelic	Gae.	Norwegian	Nor.	Welsh	Wel.

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	ml, µl	milli-, microliter(s)
ADP	adenosine diphosphate	mm	millimeter(s)
AMP	adenosine monophosphate	mOsm	milliosmolar; milliosmeter(s)
amp	ampere	msec	millisecond(s)
ATP	adenosine triphosphate	mV	millivolt(s)
A	angstrom	min	minute(s)
BSP	sulfobromophthalein	MTD	maximum tolerated dose
C	degrees centigrade	NAD	nicotinamide adenine dinucleotide
cal	calories	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter		
cm	centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
dl	deciliter		
DNA	deoxyribonucleic acid	NADPH	reduced nicotinamide adenine dinucleotidephosphate
ED_{50}	median effective dose(s)		
EDTA	ethylenediamine tetraacetic acid	ng	nanogram (10^{-9})
e.g.	for example	nm	nanometer(s)
EEG	electroencephalogram	Pa	ambient pressure
ESR	erythrocyte sedimentation rate	PAS	periodic acid - Schiff
g	gram(s)	pg	picogram (10^{-12})
µg	microgram(s)	pgEq	picogram equivalent(s)
Hz	Hertz	p.o.	orally
hr	hour(s)	ppb	parts per billion
Ig	immunoglobulin(s)	ppm	parts per million
i.m.	intramuscular	r	Roentgen
i.p.	intraperitoneal	RBC	red blood cells (erythrocytes), red blood count
IU	international unit(s)		
i.v.	intravenous	resp.	respectively
kcal	kilocalorie(s)	RNA	ribonucleic acid
kg	kilogram(s)	s.c.	subcutaneous
K_m	Michaelis constant	sec	second(s)
LD_{50}	median lethal dose(s)	SGOT	serum glutamic-oxalacetic transaminase
LDH	lactic acid dehydrogenase		
m	meter(s)	SGPT	serum glutamic-pyruvic transaminase
M	molar; mole(s)		
mM	millimolar; millimole(s)	U	unit(s)
µM	micromolar; micromole(s)	UV	ultraviolet
ma	milliampere(s)	V	volt
mCi, µCi	milli-, microcurie(s)	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mg	milligram(s)	yr	year(s)

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

7974 DISTRIBUTION OF GASTRIN CELLS IN THE GASTRIC MUCOSA: AN IMMUNOFLUORESCENCE STUDY. (Jpn.) Nagata, T. (Second Dept. Pathology, Kyushu Univ., Fukuoka 812, Japan). *Fukuoka Acta Med.* 68(7):313-326; 1977.

The morphology and distribution of gastrin cells in gastric mucosal surgical specimens from 27 patients with carcinoma, 13 with gastric ulcer, 19 with duodenal ulcer, and 4 with other lesions were examined, using an indirect immunofluorescence method. Synthetic human 1-17 gastrin antiserum was used. Gastrin cells were found almost exclusively in the mucous membrane of the pyloric antrum, and their distribution was patchy. They were most numerous in the foveolae at the level of the middle third of the mucosal layer. Smaller numbers of cells were observed in the pyloric glands, the foveolae of the fundic mucosa, and the Brunner's glands of the duodenal bulb. No gastrin cells were found in the fundic glands, the cardiac glands, or in intestinalized gastric mucosal epithelium. Gastrin cells were frequently oval in shape, and the immunofluorescence was entirely cytoplasmic and yellow-green in color with unstained nuclei. The cytoplasm was clear when stained with hematoxylin and eosin. The cells were argyrophilic but not argentaffin. The number of gastrin cells decreased with increasing age of the patient and with increased intestinal metaplasia and gastric mucosal atrophy. In cases without intestinal metaplasia, the number of gastrin cells in the gastric mucosa within 7 cm from the pyloric ring ranged from 200/cm to 250/cm; the number decreased gradually as the distance from the pyloric ring increased. There was no apparent relationship between the number of gastrin cells and gastric secretion.

7975 EVIDENCE OF INNERVATION OF THE SMALL INTESTINE FROM THE CERVICAL SYMPATHETIC GANGLIA. (Eng.) Ahlman, B. H.; Lundberg, J. M.; Dahlstrom, A.; Larsson, I.; Pettersson, G.; Kewenter, J.; *et al.* (Dept. Surgery III, Univ. Goteborg, Goteborg, Sweden). *J. Surg. Res.* 24(3):142-149; 1978.

To study further the possible nervous pathways from the sympathetic neck ganglia via the vagal nerves to the intestine of the cat, intraoperative nerve-crush procedures were combined with the fluorescence method of Hillarp-Falck to visualize monoamine-containing nerve fibers and with the horseradish peroxidase (HRP) technique to demonstrate neuronal pathways by retrograde mapping. Two normal cats and two in which the superior cervical ganglia (SCG) bilaterally had been surgically removed were subjected to crushing of the right vagal nerve at the cervical and subdiaphragmatic levels 30-45 min before this vagal tissue was dissected for examination by fluorescence microscopy. Four other cats were injected at several hundred 0.5-cm intervals around the circumference of the gut wall with HRP (0.5-1.0 g/animal). Unilateral (right) crush of the cervical vagus and the cervical sympathetic chain was performed immediately (cat I), 24 hr after the injections (cats II and III), or not at all (cat IV); the tissues were dissected for examination at 24, 60, 72 hr, or 5 days, resp., in cats I-IV. In the two normal cats with intact SCG, many fibers with accumulated fluorescent material were seen cranial to the crush at the cervical level, due to a proximodistal, intra-axonal transport of amine storage granules accumulating proximal to the crush site. At the subdiaphragmatic level, the vagal nerve contained a large number of fluorescent fibers cranial to the crush. Removal of the SCG caused a complete disappearance of the fluorescent fibers at the cervical level, but many fluorescent fibers still remained at the subdiaphragmatic level. In all the cats, the celiac mesenteric inferior ganglion contained many HRP-positive cells. No other region in cat I was HRP-positive. In cats II and III, a few HRP-positive cells were seen in the left nodose ganglion, left SCG, and left and right stellate ganglia. In cat IV, all examined ganglia contained (on both sides) either a few HRP-positive cells (in the thoracic, nodose, and superior cervical ganglia) or a moderate number of HRP-positive cells (in the stellate ganglia). The fluorescence studies demonstrate that in the cat the cervical vagus carries catecholamine (CA)-containing fibers, emanating from the SCG. The persistence of CA fibers at the subdiaphragmatic level demonstrates that the vagus nerve receives more CA fibers caudal to the cervical level, probably from the stellate, and possibly from the intrathoracic sympathetic ganglia. The results clearly demonstrate that not only the stellate ganglia but also the SCG via the vagus nerve send CA fibers that innervate part of the small intestine, strongly modifying the classical hypothesis that these ganglia innervate mainly the head, neck, and heart.

7976 TOPOGRAPHIC STUDY OF THE SMALL INTESTINAL EPITHELIAL CELL SURFACE AND ADAPTIVE CHANGES INDUCED BY PROXIMAL RESECTION [Abstract]. (Eng.) Freeman, H.; Garrido, A.; Kim, Y.; Etzler, M. (Veterans Admin Hosp., San Francisco, CA). *Gastroenterology* 72(5, Part 2):1059; 1977.

7977 ULTRASTRUCTURE OF THE GUT NEUROTENSIN CELL [Abstract]. (Eng.) Alumets, J.; Sundler, F.; Hakanson, R. (Dept. Histology, Univ. Lund, Lund, Sweden). *Acta Endocrinol. (Kbh.)* 85 (Suppl. 212):104; 1977.

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

7978 THE RIGHT BRANCHES OF THE SUPERIOR MESENTERIC ARTERY FOR THE VASCULARIZATION OF THE LARGE BOWEL [Abstract]. (Eng.) Ilic, A.; Blagotic, M.; Marinkovic, S. (Medical Faculty, Belgrade, Yugoslavia). *Acta Anat. (Basel)* 99(1): 96-97; 1977.

7979 INNOMINATE LINES OF THE COLON. RADIOLOGICAL-HISTOLOGICAL CORRELATION. (Eng.) Matsuura, K.; Nakata, H.; Takeda, N.; Nakata, S.; Shimoda, Y. (Kyushu Univ., Sch. Medicine, Maidashi, Higashi-Ku, Fukuoka-Shi, Japan). *Radiology* 123(3): 581-584; 1977.

7980 THE MORPHOLOGY OF THE FIBROUS TISSUE OF SUBMUCOSA OF THE LARGE INTESTINE AND THE EFFECT OF VARIOUS SUTURE MATERIALS [Abstract]. (Eng.) Lord, M. G.; Broughton, A. C.; Williams, H. T. G. (Manchester Royal Infirmary, Oxford Rd., Manchester 13, England). *Eur. Surg. Res.* 9(Suppl. 1): 161; 1977.

7981 MORPHOLOGIC ALTERATIONS IN RATS WITH CHRONIC ENDOGENOUS HYPERGASTRINEMIA [Abstract]. (Eng.) Owen, R. L.; Deveney, K.; Way, L. W. (Veterans Admin. Hosp., San Francisco, CA). *Clin. Res.* 26(2):112A; 1978.

7982 ADENYLATE CYCLASE IS THE BEST MARKER OF THE PLASMA MEMBRANE OF HUMAN HEPATOCYTE [Abstract]. (Eng.) Duvaldestin, P.; Pecker, F.; Hanoune, J.; Berthelot, P. (Unite de Recherches INSERM U-99, Hopital Henri Mondor, Creteil, France). *Digestion* 16(4):337-338; 1977.

7983 ESOPHAGEAL LENGTHS IN CHILDREN: CORRELATION WITH AGE, SURFACE AREA AND HEIGHT [Abstract]. (Eng.) Strobel, C. T.; Byrne, W. J.; Euler, A. R.; Ament, M. E. (Dept. Pediatrics, Univ. California, Los Angeles, CA). *Clin. Res.* 26(2): 174A; 1978.

7984 QUANTITATION OF INTESTINAL MUCOSAL SURFACE AREA BY STEREOLOGY [Abstract]. (Eng.) Heneghan, J. P.; Heneghan, J. B. (Louisiana State Univ. Sch. Medicine, New Orleans, LA 70112). *Physiologist* 20(4):42; 1977.

See also, 8019, 8020, 8073, 8121, 8125, 8126, 8128, 8138, 8198, 8327, 8353, 8518, 8567.

ABSORPTION - EXCRETION - TRANSPORT

7985 THE BILE DUCT LIGATED ANIMAL: A MODEL FOR DETERMINING TOTAL INTESTINAL ABSORPTION. (Eng.) Halladay, S. C.; Enderlin, F. E.; Parkinson, T. M.; Honohan, T. (Biological Sciences, Dynapol, 1454 Page Mill Rd., Palo Alto, CA 94034). *Drug Chem. Toxicol.* 1(2):203-218; 1978.

A new bile duct-ligated animal model for determining total intestinal absorption was evaluated using compounds with markedly different metabolism and excretion characteristics. The test solutions were administered p.o. by gavage in control and bile duct-ligated female Sprague-Dawley rats or ICR/Sim mice. A comparison of the serum concentration time curves for digoxin, bumetanide, imipramine, hexobarbital, and sodium benzoate in ligated and control rats indicated that bile duct ligation had no significant effect on absorption *per se* during the study period. In ligated rats, total absorption of imipramine (which undergoes enterohepatic circulation), as estimated from urine and tissue recoveries, was 92%. A significant overestimation of total absorption (128%) resulted from combining urine and tissue recoveries from controls with bile recoveries from cannulated rats. For sodium benzoate, which undergoes little or no enterohepatic circulation, there was no significant difference in total recoveries determined by these two methods (97% versus 101%). In ligated mice, absorption of imipramine and sodium benzoate was 80% and 89%, resp. This model simply and accurately estimates intestinal absorption of both types of compounds, and eliminates the error inherent in determining absorption of compounds that are excreted in the bile when urine, feces, and tissue data from one animal are combined with bile data from another. The bile duct-ligated animal model is applicable especially to small animals such as mice, in which bile duct cannulation is extremely difficult, and to the study of compounds whose properties do not permit traditional methods to be used.

7986 EFFECT OF MODIFIED SHAM FEEDING ON JEJUNAL TRANSPORT AND PANCREATIC AND BILIARY SECRETION IN MAN. (Eng.) Read, N. W.; Cooper, K.; Fordtran, J. S. (Univ. Texas Health Science Center, Dallas, TX 75235). *Am. J. Physiol.* 234 (4):E417-E420; 1978.

The effect of modified sham feeding (thoroughly masticating but not swallowing an appetizing meal) on the intestinal transport of water and electrolytes was studied using jejunal perfusion via a triple-lumen tube to investigate whether intes-

tinal secretion is under parasympathetic control. Ten volunteers who had experienced intestinal perfusion previously and of whom three had pentagastrin-fast achlorhydria (controls for acid effect) were used in the 4-hr experiment (the 1st, 3rd, and 4th hr were control periods, the 2nd hr was the experimental sham feeding). Gastric perfusion was continuous; the infusion site was the duodenojejunal flexure, and the collecting site was the gastric antrum; measurements were performed to assure that no gastric acid entered the test segment. Sham feeding produced a significant rise ($p<0.01$) in gastric acid output in the seven normal subjects compared with the achlorhydric controls. Movement of water and electrolytes across the jejunal wall and the jejunal potential difference made no significant response to sham feeding. There was no consistent change in the output of amylase, protein, or bile acids in response to sham feeding in the normal or achlorhydric subjects. The results suggest that cephalic stimulation of the vagus by itself does not influence jejunal transport, but the vagus may act in concert with other factors as a regulator of jejunal transport. The results also suggest that fluctuation in bile acid secretion is not an important regulator of ion transport in the jejunum, at least in the basal state.

7987 INTESTINAL ABSORPTION OF ASCORBIC ACID AND ITS PASSAGE THROUGH THE LIPOID MEMBRANE OF GUINEA PIGS. (Eng.) Odumosu, A.; Wilson, C. W. M. (Dept. Pharmacology, Trinity Coll., Dublin 2, Ireland). *Int. J. Vitam. Nutr. Res.* 48 (1):3-11; 1978.

The intestinal absorption of ascorbic acid (AA) and its passage through the lipoid membrane was studied in normal (N), hypervitaminoic (HV), and scorbutic (S) guinea pigs. Measurements were made in ligated segments of the intestinal lumen of intact living rats. The animals received either a scorbutogenic diet alone (group S) or this diet plus 20 mg vitamin C/day (in group N) or 100 mg vitamin C/day (in group HV) for 27 days prior to the absorption study. Plasma and WBC concentrations of AA were measured before and after AA (20 mg in 1 ml) or saline (1 ml) had been introduced into the intestinal loops where the solutions were then maintained for 2 hr. After introduction of AA, small and insignificant elevations in the WBC AA concentrations occurred in each group, and the plasma levels of AA rose from 0.60 ± 0.3 to 0.96 ± 0.07 mg/ml ($p<0.02$) in the N group and from 1.14 ± 0.18 to 1.57 ± 0.17 mg/ml ($p<0.05$) in the HV group, but only from 0.11 ± 0.01 to 0.13 ± 0.09 mg/ml in the S group. When the retention of AA in the intestinal loops was measured following saline and AA treatments for 2 hr, the largest amount of AA remained in the loops of the S group (8.38 mg/ml) compared with the N group (2.9 mg/ml) and the HV group (3.85 mg/ml), the S group value being significantly different from the other two ($p<0.02$). Mucosal cell uptake of AA was least in the HV group (3.68 mg/g weight), greater in the N group (7.12 mg/g), and greatest in the S group (10.13 mg/g), all differences being significant ($p<0.01$ or $p<0.05$). The muscle content of AA was highest in the S group after the introduction of AA (1.94 mg/g weight) compared with the N and the HV groups (1.75 mg/g in both). Preferential intestinal cell absorption of AA in the S guinea pigs was demonstrated: the AA uptake from the intestinal lumen and retention in the mucosal cells was greatest in this group, but impaired transfer of AA to the plasma also occurred. The S mucosal cells showed histological signs of damage, which may account for this negligible transfer of AA from the intestine into plasma. The high AA concentration in S mucosal cells may be due to a passive diffusion mechanism resulting from the relatively high concentration of AA in the intestinal lumen. It is possible that in acute scurvy, only passive transfer of AA occurs, while active transfer into the plasma is suppressed.

7988 ROLE OF SULFHYDRYL GROUPS IN ACID TRANSPORT BY STOMACH. (Eng.) Fromm, D.; Fuhro, R. (Harvard Medical Sch., Boston, MA). *Surg. Forum* 28:351-353; 1977.

The effects of two sulfhydryl-reactive agents, *p*-chloromercuribenzene sulfonic acid (PCMBS) and dithiothreitol (DTT), on acid secretion and backward diffusion by isolated rabbit gastric mucosa were studied. The spontaneous acid secretory rate of fundic mucosa (2.0 ± 0.2 μEq/hr cm^2) was constant over 90 min. Addition of sulfhydryl-oxidizing PCMBS (0.5 mM) to the mucosal side significantly decreased the basal rate of acid secretion to 0.2 ± 0.1 μEq/hr/cm^2 ($p<0.001$), but its addition to the serosal side was without effect. Addition of sulfhydryl-reducing DTT (4 mM) to the mucosal side significantly stimulated acid secretion to 2.7 ± 0.3 μEq/hr/cm^2 ($p<0.001$) in both the absence and presence of PCMBS whose inhibitory effect DTT was able to prevent and reverse. Addition of 4 mM cysteine (another sulfhydryl-reducing agent) to the mucosal side did not stimulate acid secretion but did completely prevent inhibition by PCMBS. The presence of PCMBS increased the rate of luminal acid loss by the fundic mucosa (from 0.3 ± 0.1 to 1.3 ± 0.2 μEq/hr/cm^2, $p<0.001$) and decreased the rate of luminal acid loss by antral mucosa (from 1.0 ± 0.2 to 0.4 ± 0.1 μEq/hr/cm^2, $p<0.001$). DTT alone did not change luminal acid loss by the antrum; it reversed the PCMBS effect on luminal acid loss by the fundus. The mean difference between the mucosal and serosal rates of CO_2 appearance approximated the rate of acid secretion both before and after PCMBS exposure (2.8 ± 0.5 and 0.2 ± 0.1 μM/hr/cm^2, resp.) but responded to DTT by increasing significantly (from 2.2 ± 0.6 to 3.3 ± 0.6 μM/hr/cm^2, $p<0.025$), indicating that both agents primarily affect mineral rather than organic acid secretion. Both mucosal and serosal addition of PCMBS caused a 52-85% decrease in shortcircuit current ($p<0.001$) and a 15-35% increase in tissue electrical resistance of the fundus ($p<0.001$). DTT alone did not affect these parameters but did reverse the effects of PCMBS on them. It is concluded that the sulfhydryl sites associated with acid secretion are located on the luminal side of the mucosa, that they

are separate from the sites associated with active transport of sodium and chloride, that the increase in backward diffusion caused by PCMBS is due to inhibition of acid secretion, and that the stimulatory effect of DTT alone is not due to the prevention of spontaneous oxidation of sulfhydryl groups.

7989 D-XYLOSE ABSORPTION TEST: PHARMACOKINETIC AND STATISTICAL STUDY. (Fre.) Huguenin, P.; Cochet, B.; Balant, L.; Loizeau, E. (Hopital Cantonal, CH-1211 Geneve 4, Switzerland). *Schweiz. Med. Wochenschr.* 108(6):206-214; 1978.

D-xylose pharmacokinetics were studied in six healthy subjects by serial measurement of blood and urinary D-xylose levels following p.o. or i.v. administration of D-xylose. Four tests were used in these subjects: A--23 g D-xylose in 1,000 ml 0.45% NaCl, i.v., over 60 min; B--4.6 g D-xylose in 1,000 ml 0.45% NaCl, i.v., over 60 min; C--25 g D-xylose in 500 ml H_2O, p.o., over 5 min; and D--5 g D-xylose in 500 ml H_2O, p.o., over 5 min. NaCl (0.9%, 500 ml) was perfused over 60 min during tests C and D. A different order of the tests was used for each subject; there was a 1-week or 1-month interval between tests. In addition, 13 patients with obesity, renal, or hepatic insufficiency or a T-tube after cholecystectomy were also investigated. Following i.v. administration of D-xylose, there were individual variations in both the rate and completeness of D-xylose absorption, so that the time and value of peak D-xylose blood level were highly variable. The urinary elimination was about 50% of the administered dose and was nearly complete by 8 hr. After p.o. administration, the D-xylose blood levels rose rapidly over a roughly 2-hr period (there were significant increases, $p<0.01$ or $p<0.05$, in level during this early period) and then decreased semilogarithmically over the next 6 hr. The urinary elimination was about 30% for the 25-g dose and about 40% of the 5-g dose at 8 hr. Renal insufficiency increased the apparent elimination half-life of D-xylose and markedly reduced renal D-xylose excretion. The results indicate that there is a very wide range of blood and urinary levels obtained by the D-xylose tolerance test. Urinary D-xylose excretion alone is not a reliable index of intestinal absorption.

7990 INTESTINAL APOPROTEINS DURING FAT ABSORPTION. (Eng.) Schonfeld, G.; Bell, E.; Alpers, D. H. (Washington Univ. Sch. Medicine, St. Louis, MO 63110). *J. Clin. Invest.* 61(6):1539-1550; 1978.

Apolipoprotein (Apo) A-I, ApoB, and ApoE (or arginine-rich apoprotein, ARP) were localized in rat intestinal mucosa by an indirect immunofluorescence method to compare their roles in the intracellular production of intestinal chylomicrons (and/or very low density lipoproteins, VLDL). In addition, tissue levels of ApoA-I and ApoB were measured by radioimmunoassay during fat absorption. Antisera were produced using ApoA-I isolated from rat plasma high-density lipoprotein, and ApoB and ARP from plasma VLDL by column chromatography. The apoproteins yielded single bands on polyacrylamide disc gel electrophoresis in urea and in sodium dodecyl sulfate. Anti-apoprotein antisera produced in rabbits appeared to be monospecific on double-antibody immunoprecipitation of ^{125}I-labeled apoproteins. In fasted animals, granular staining of ApoA-I was noted in the Golgi regions of epithelial cells in the top third of the villus. At 30 min, when fat droplets were seen in the supranuclear cytoplasm of the cells along the top two-thirds of the villus, intense ApoA-I staining surrounded the cytoplasmic droplets. Later, when epithelial cells and lamina propria both contained fat droplets, bright ApoA-I stain surrounded many droplets in the supranuclear cytoplasm and in the lamina propria. Over the same period, ApoA-I tissue levels rose 10-fold. The distribution and time-course of ApoB staining was nearly identical with that of ApoA-I. Concomitantly, tissue ApoB levels doubled. In contrast, in fasting rat intestine, ARP staining was sparse, punctate, and confined to the lower quarter of the villus. After fat feeding, stained droplets were seen only in the lamina propria near the base of the villus, even though abundant ARP occurred in cells along most of this length of the villus. Stain never surrounded droplets inside cells. Thus, ApoA-I and ApoB appear to participate in the intracellular assembly of gut lipoproteins, whereas ARP does not, although ARP was found within the mucosal cells. The liver stained heavily for ARP and lightly for ApoA-I, whereas intestine stained heavily for ApoA-I and lightly for ARP. Both organs stained for ApoB. These findings suggest that there may be some quantitative "specialization" of the two organs that secrete lipoproteins.

7991 ENTERAL ABSORPTION OF HIGH CALCIUM DOSES, AS DETERMINED BY MEANS OF STABLE ISOTOPES. (Ger.) Behne, D.; Bratter, P.; Herzfeld, U.; Kraft, D. (Bereich Kernchemie und Reaktor, Hahn-Meitner-Institut fur Kernforschung Berlin, Glienicker Str. 100, D-1000 Berlin 39, W. Germany). *Klin. Wochenschr.* 56(2):69-74; 1978.

The enteral resorption of high p.o. Ca doses (500 and 1,000 mg; calcium-Sandoz forte and calcium-Sandoz fortissimum, resp.) was determined in eight healthy subjects by use of a double-isotope method. The isotopes were assayed by neutron activation analysis. The stable ^{48}Ca isotope was used to label the p.o. Ca dose. The rapidly exchangeable body Ca pool was measured by the i.v. administration of $CaCl_2$ enriched with the stable ^{46}Ca isotope immediately after p.o. administration of the p.o. Ca doses. The 24-hr Ca exchange, determined from the serum ^{46}Ca level, was found to be 6.4 ± 1 g or 98.8 ± 15.4 mg/kg of body weight. For the p.o. administered Ca, the elimination constant was 0.980 for the 500-mg Ca dose, and 0.880 mg for the 1,000-mg dose. The invasion constant was 0.056 and 0.073, resp., for the two doses while the elimination half-life was 185 min for either dose. The resorption rate was 30.5% for 500 mg

and 28% for 1,000 mg, i.e., the resorption rate was independent of the dose administered. The serum Ca level showed a short-term rise following the oral administration of 1,000 mg Ca. The findings indicate that the quantity of resorbed Ca is directly proportional to the p.o. dose at high doses and that high p.o. Ca doses can be used therapeutically in degenerative bone diseases.

7992 THE GASTROINTESTINAL ABSORPTION OF LIPOSOMALLY ENTRAPPED INSULIN IN NORMAL RATS [Abstract]. (Eng.) Patel, H. M.; Ryman, B. E. (Charing Cross Hosp. Medical Sch., Fulham Palace Rd., London W6 8RF, England). *Biochem. Soc. Trans.* 5(4):1054-1055; 1977.

7993 INTESTINAL ABSORPTION OF BOTULINUM TOXINS OF DIFFERENT MOLECULAR SIZES IN RATS. (Eng.) Sugii, S.; Ohishi, I.; Sakaguchi, G. (Coll. Agriculture, Univ. Osaka Prefecture, Sakai-shi, Osaka 591, Japan). *Infect. Immun.* 17(3):491-496; 1977.

7994 MECHANISMS INVOLVED IN TRANSCAPILLARY FLUID MOVEMENT INTO THE SECRETING CAT SUBMANDIBULAR GLAND [Abstract]. (Eng.) Lundvall, J.; Holmberg, J. (Dept. Physiology, Univ. Lund, Lund, Sweden). *Acta Physiol. Scand.* 102(1):16A; 1978.

7995 ABSORPTION OF PANCREATIC LIPASE BY THE DUODENUM INTO LYMPH VESSELS [Abstract]. (Eng.) Papp, M.; Feher, S.; Folly, G.; Horvath, E. J. (Inst. Experimental Medicine, Hungarian Acad. Sciences, H-1450 Budapest, Hungary). *Ir. J. Med. Sci.* 146(Suppl. 1):23-24; 1977.

7996 PLACENTAL TRANSFER OF BILE PIGMENTS IN RATS [Abstract]. (Eng.) Palma, L.; McDonagh, A. F. (Liver Center, Univ. California, San Francisco, CA 94143). *Gastroenterology* 73(5): 1237; 1977.

7997 EFFECT OF BILIARY LIPIDS ON THE ADSORPTION OF PANCREATIC LIPASE AND COLIPASE AT INTERFACES [Abstract]. (Eng.) Lairon, D.; Leonardi, J.; Nalbone, G.; Lafont, H.; Domingo, N.; Hauton, J. C.; Verger, (INSERM U-130, Group de Recherches sur le Transport des Lipides, 46 Bd. de la Gaye, 13009 Marseille, France). *Ir. J. Med. Sci.* 146 (Suppl. 1):11; 1977.

7998 EFFECT OF LUMINAL HYPEROSMOLALITY ON H^+ LOSS IN DUODENUM [Abstract]. (Eng.) Johnson, G. M.; Chung, R. K. (Veterans Admin. Hosp., Iowa City, IA 52240). *Fed. Proc.* 37(3):698; 1978.

7999 JEJUNAL ABSORPTION OF GLUCOSE IN RATS CHRONICALLY INFECTED WITH Trypanosoma cruzi. (Eng.) Salgado, J. A.; de P. Castro, L.; Ribeiro, T. C.; Pessoa, W.; de Oliveira, J. P. M.; Bambirra, E. A.; *et al.* (Faculdade de Medicina, Universidade Federal de Minas Gerais, Caixa Gerais, Caixa Postal, 340, 30000 Belo Horizonte, MG., Brazil). *Arq. Gastroenterol.* 14(4):210-214; 1977.

8000 MODIFICATION OF TRANSPORT PROCESSES ACROSS RAT INTESTINE [Abstract]. (Eng.) Hardcastle, J.; Hardcastle, P. T.; Sandford, P. A. (Dept. Physiology, Univ. Sheffield, Sheffield, England). *Br. J. Pharmacol.* 62(3):463P; 1978.

8001 TRANSPORT CHARACTERISTICS OF ISOLATED NEWBORN RABBIT ILEUM. (Eng.) Cooke, H. J.; Dawson, D. C. (Kansas Univ. Medical Center, Kansas City, KS 66103). *Am. J. Physiol.* 234(3): E257-E260; 1978.

8002 INTESTINAL TRANSPORT OF SALT AND WATER. (Eng.) Turnberg, L. A. (Hope Hosp., Eccles Old Rd., Salford M6 8HD, Lancashire, England). *Clin. Sci. Mol. Med.* 54(5):337-348; 1978.

8003 LANTHANUM AS A TOOL TO STUDY THE ROLE OF PHOSPHATIDYLINOSITOL IN THE CALCIUM TRANSPORT IN RAT PAROTID GLANDS UPON CHOLINERGIC STIMULATION. (Eng.) Keryer, G.; Rossignol, B. (Institut de Biochimie, Universite de Paris-Sud, Batiment 432, F-91405 Orsay-Cedex, France). *Eur. J. Biochem.* 85(1):77-83; 1978.

8004 CHANGES IN ABSORPTION OF GLUCOSE AND PROLINE FOLLOWING IRRADIATION TO THE EXTERIORIZED ILEUM. (Eng.) Mohiuddin, M.; Tamura, K.; DeMare, P. (Thomas Jefferson Univ. Hosp., Philadelphia, PA 19107). *Radiat. Res.* 74(1): 186-190; 1978.

See also, 8022, 8130, 8135, 8156, 8210, 8309, 8312, 8315, 8570.

MOTILITY

8005 THE EFFECTS OF PARAOESOPHAGEAL STRUCTURES AND VAGOTOMY ON THE CANINE LOWER OESOPHAGEAL SPHINCTER FUNCTION. (Eng.) Laitinen, S. (Univ. Central Hosp., SF-90220 Oulu 22, Finland). *Ann. Chir. Gynaecol.* 66(6):304-310; 1977.

The effects of extrinsic paraesophageal structures and of the vagus nerve on lower esophageal function were investigated in dogs. Manometry demonstrated a high-pressure zone at the gastroesophageal junction of all 25 dogs examined. In

all dogs in which the gastroesophageal junction was isolated from surrounding structures by a stiff-walled Silastic tube, the mean resting lower esophageal sphincter pressure (LESP) decreased from a preoperative level of 14.0 ± 3.0 mm Hg to 13.0 ± 2.7 mm Hg. Transabdominal subdiaphragmatic vagotomy, transthoracic subhilar vagotomy, or cervical vagotomy did not significantly change the mean resting LES pressure, but the response to abdominal compression was significantly decreased ($p<0.01$) by all three procedures. The vagus nerves apparently have no major role in the maintenance of resting LESP. The LES response to increased intra-abdominal pressure seems to represent a genuine increase in LES tone, to which extrinsic mechanical factors add only a small contribution. This response seems to be mediated by a vagal reflex arc, the afferent part of which has its origin below the diaphragm.

8006 RELATION OF GASTROESOPHAGEAL SPHINCTER PRESSURE AND ESOPHAGEAL CONTRACTILE WAVES TO AGE IN MAN. (Eng.) Csendes, A.; Guiraldes, E.; Bancalari, A.; Braghetto, I.; Ayala, M. (Hospital J. J. Aguirre, Santos Dumont 999, Santiago, Chile). *Scand. J. Gastroenterol.* 13(4):443-447; 1978.

To determine a normal range of values for gastroesophageal sphincter pressure (GESP) and peristaltic wave amplitude (PWA) and variations in these levels among subjects of different age groups, esophageal motility was studied in 113 normal subjects ranging in age from 2 months to 74 yr. The GESP levels were significantly higher in children aged 2-5 months (37.2 ± 3.8 mm Hg) than in all age groups over 1 yr of age ($p<0.001$). With increasing age, the resting GESP gradually fell to a mean of slightly over 12 mm Hg in age groups between 15 and 24 yr and between 55 and 64 yr. There was a further drop in subjects aged 65 to 74 yr. Children in the 6- to 12-month age group also had significantly higher ($p<0.01$) pressures than did all adult groups and children in the 6- to 14-yr age group, but not from children in the 1- to 5-yr age group. In the adult population, the lowest GESP recorded was 7 mm Hg and the highest 20 mm Hg. Sphincter length increased with age from 1.1 cm in the 2- to 5-month age group up to 2.4 cm in the 15- to 24-yr group. There was no further change thereafter. The PWA was similar in all age groups except those 65 yr or older; in the latter group, the percentage of peristaltic waves induced by deglutition was 75%, while that in all other groups ranged between 82% and 100%.

8007 THE ROLE OF LOCAL CHOLINERGIC PATHWAYS IN THE MOTOR RESPONSE TO CHOLECYSTOKININ AND GASTRIN IN ISOLATED GUINEA-PIG FUNDUS AND ANTRUM. (Eng.) Gerner, T.; Haffner, J. F. W. (Ullevål Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(6):751-757; 1977.

Studies were carried out to determine to what extent the local cholinergic pathways participate in the motor response to cholecystokinin-pancreozymin (CCK-PZ) and gastrin in guinea pig fundus and antrum *in vitro*. The motor responses to acetylcholine and the ganglion-stimulating agent, dimethylphenylpiperazinium (DMPP), were studied for comparison. CCK-PZ (0.38 U/ml), gastrin (0.05 µg/ml), and acetylcholine (10^{-3} M) increased antral rhythmic activity and fundal basal pressure ($p<0.05$). DMPP (10^{-5} M) also generally increased antral rhythmic activity, but it invariably induced a drop in fundic pressure. The antral motor effects were significantly ($p<0.05$) blocked by 10^{-5} M atropine or 10^{-6} M tetrodotoxin. However, the fundal motor responses were almost completely unaffected. It is concluded that CCK-PZ and gastrin exert their antral motor effects essentially through local cholinergic neural pathways, while the motor responses in the fundus are almost entirely independent of these pathways.

8008 EFFECTS OF CERULEIN ON INTESTINAL MOTILITY. (Jpn.) Nakamura, N.; Shiozaki, S.; Kojima, T.; Shimizu, M.; Tanaka, M. (Pharmaceuticals Res. Lab., Kyowa Hakko Kogyo Co., Ltd., Shimotogari, Nagaizumicho, Shizuoka 411, Japan). *Folia Pharmacol. Jpn.* 73(7):743-756; 1977.

The effects of cerulein on intestinal motility were compared with those of neostigmine, pantethine, prostaglandin E_1 (PGE_1), and prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$). Cerulein stimulated electric discharges in the intestinal tracts of anesthetized rabbits and was more potent than either neostigmine or pantethine. The small intestine was more sensitive to this agent than was the large intestine. PGE_1 inhibited, while $PGF_{2\alpha}$ stimulated electrical discharges in the small intestine. Atropine did not completely inhibit the stimulatory effect of cerulein. Cerulein promoted the transit of a charcoal meal through the intestine of the mouse and was approximately 30 times more potent than neostigmine. At high doses, the promotion was reduced, and, the reduction was inhibited by reserpine or phentolamine-propranolol. These observations indicate that cerulein produces a catecholamine release at high doses. Cerulein increased intestinal transit time in cecectomized mice at doses 30 times higher than given to intact mice. Cerulein, neostigmine, PGE_1, and $PGF_{2\alpha}$ produced an excitatory effect on the isolated rabbit intestine. The minimal effective concentration of these four substances were 3×10^{-10} to 10^{-9}, 10^{-8}, 10^{-9}, and 3×10^{-11} g/ml in the ileum and 3×10^{-9} to 10^{-8}, 10^{-8} to 3×10^{-8}, 10^{-10}, and 10^{-11} g/ml in the proximal colon, resp.

8009 FACTORS INFLUENCING TONE IN RABBIT LARGE INTESTINE. (Eng.) McKirdy, H. C. (Vale of Leven District General Hosp., Alexandria, Dunbartonshire, Scotland). *Q. J. Exp. Physiol.* 63(2): 111-118; 1978.

8010 ROLE OF GASTRIC EMPTYING ON ETHANOL POISONING IN RATS. (Eng.) Hillbom, M. E.; Wallgren, H. (Res. Lab., State Alcohol Monopoly, Box 350, SF-00101 Helsinki 10, Finland). *Med. Biol.* 56(1):37-41; 1978.

8011 A SIMPLE DEVICE MEASURING DIFFERENCES IN LEVEL IN THE OESOPHAGUS. (Eng.) Ask, P.; Tibbling, L. (Univ. Hosp., S-581 85 Linkoping, Sweden). *Acta Otolaryngol.* 85(3/4):296-297; 1978.

8012 SIMULTANEOUS RECORDING OF GASTRIC AND DUODENAL ELECTRICAL ACTIVITY. (Eng.) Civalero, L. A.; Kantelius, M.; Nilsson, B. Y. (Sodersjukhuset, S-100 64 Stockholm, Sweden). *Scand. J. Gastroenterol.* 13(4):459-463; 1978.

8013 THE EFFECT OF PAPAVERINE ON THE CONTRACTION OF THE HUMAN GALLBLADDER. (Eng.) Katevuo, K.; Kanto, J.; Manell, D.; Mantyla, R. (Dept. Radiology, Turku Univ., Turku, Finland). *Int. J. Clin. Pharmacol. Biopharmacol.* 16(5):220-222; 1978.

8014 NEURAL CONTROL OF PRIMARY ESOPHAGEAL PERISTALSIS [Letter to Editor]. (Eng.) Janssens, J.; Vantrappen, G.; Hellemans, J.; Diamant, N. E.; El-Sharkawy, T. Y. (Dept. Medical Res., Univ. Leuven, Leuven, Belgium). *Gastroenterology* 74(4):801-803; 1978.

8015 THE ADRENERGIC INNERVATION OF THE SUBMANDIBULAR GLAND OF THE CAT AND THE EFFECTS OF VARIOUS SURGICAL DENERVATIONS ON THESE NERVES: A HISTOCHEMICAL AND ULTRASTRUCTURAL STUDY INCLUDING THE USE OF 5-HYDROXYDOPAMINE. (Eng.) Garrett, J. R.; Kemplay, S. K. (King's Coll. Hosp. Dental Sch., London SE5 8RX, England). *J. Anat.* 124(1):99-115; 1977.

See also, 8016, 8017, 8053, 8114, 8123, 8185, 8262, 8270, 8274, 8275.

SECRETION AND METABOLISM

8016 SECRETORY, MOTOR AND VASCULAR EFFECTS IN THE SUBLINGUAL GLAND OF THE RAT CAUSED BY AUTONOMIC NERVE STIMULATION. (Eng.) Templeton, D.; Thulin, A. (Inst. Physiology, Univ. Lund, Lund, Sweden). *Q. J. Exp. Physiol.* 63(1):59-66; 1978.

8017 THE ENDOCRINE CONTROL OF GASTROINTESTINAL FUNCTION. (Eng.) Chey, W. Y.; Gutierrez, J. C. (Genesee Hosp., Rochester, NY). *Adv. Intern. Med.* 23:61-84; 1978.

8018 DEVELOPMENT OF SECRETORY FUNCTION IN RAT PAROTID GLAND. (Eng.) Grand, R. J.; Schay, M. I. (Children's Hosp. Medical Center, 300 Longwood Ave., Boston, MA 02115). *Pediatr. Res.* 12(2):100-104; 1978.

8019 CELLULAR LOCALISATION OF HUMAN UROGASTRONE/EPIDERMAL GROWTH FACTOR. (Eng.) Elder, J. B.; Williams, G.; Lacey, E.; Gregory, H. (Dept. Surgery, Univ. Manchester, Manchester, England). *Nature* 271(5644):466-467; 1978.

See also, 7981, 7994, 8003, 8130.

SECRETION AND METABOLISM

Stomach

8020 ULTRASTRUCTURAL CHANGES IN OXYNTIC CELLS ASSOCIATED WITH SECRETORY FUNCTION: A MEMBRANE-RECYCLING HYPOTHESIS. (Eng.) Forte, T. M.; Machen, T. E.; Forte, J. G. (Donner Lab., Univ. California, Berkeley, CA 94720). *Gastroenterology* 73(4, Part 2):941-955; 1977.

Changes in piglet oxyntic cells associated with secretory function were studied ultrastructurally after the induction of secretion by histamine and after removal of the secretagogue. Morphological changes consisting chiefly of proliferation of the secretory surface were detected in the piglet

oxyntic cell as early as 3 min after the addition of 10^{-5} M histamine to the preparation and before the production of detectable amounts of HCl secretion. However, there were frank changes in electrophysiology indicative of secretion: mucosal resistance and potential difference decreased. Microfilaments in the resting state and in the early phases of the onset of HCl secretion formed a ring of parallel filaments just beneath the microvillar plasma membrane; during maximal secretion, there appeared to be some detachment of microfilaments, which then appeared condensed in the center of the elongated microvillar processes. During the early stages of secretagogue withdrawal, when microvilli were condensed, the microfilaments showed great disorganization. With reestablishment of the resting state, the microfilaments were again aligned parallel with the microvilli. Structural changes associated with the reestablishment of the resting state were striking. In general, the apical surface membrane was reorganized or recycled back into the cytoplasmic tubulovesicular form. Analysis of intervening structural events showed a complex series of membrane alterations, which were characterized as: (a) condensation of elongated apical microvilli within the canalicular space; (b) close apposition and association of secretory membrane surfaces; (c) massive endocytosis of surface membranes into the cytoplasm, frequently in the form of pentalaminar membrane structures; and (d) reappearance of tubulovesicle profiles within the cytoplasm. The endocytosed pentalaminar structures had a thickness (242 A) just double that of single plasma membranes (119 A), thus making it unlikely that they represent fused membranes. Still, the very close apposition and contact of these membrane surfaces suggest that critical physical changes underlie this phase of membrane uptake. Morphological events after the withdrawal of histamine suggest that there must be important controlling steps in the phase of massive membrane uptake and in the reformation of tubulovesicular structures.

8021 ADENYLATE CYCLASE OF HUMAN GASTRIC MUCOSA. (Eng.) Simon, B.; Kather, H. (Medizinische Universitatsklinik Heidelberg, Bergheimerstrasse 58, D-6900 Heidelberg, W. Germany). *Digestion* 16(1/2):175-179; 1977.

Adenylate cyclase activity was studied in homogenates of human gastric mucosa, and the effects of histamine and catecholamines on this activity were examined. Stomach tissue was obtained by subtotal gastric resection from seven patients with peptic ulcer. The mean basal adenylate cyclase activity of the gastric mucosa averaged 0.85 ± 0.4 nmol cyclic AMP (cAMP) formed/mg protein/15 min. Saturating concentrations of histamine (5×10^{-3} M) caused an increase in enzyme activity of about fourfold. cAMP accumulation in the same preparation was enhanced approximately two- to threefold by maximally effective concentrations of epinephrine (5×10^{-4} M). The concentrations required for half maximal stimulation were 5×10^{-5} M for both activators. Isoproterenol stimulated the enzyme activity to about the same degree as did epinephrine. The stimulatory action of catecholamines and histamine were additive, indicating that these substances act on separate adenylate cyclases. The histamine responsiveness of the enzyme system was almost completely abolished by 1×10^{-3} M cimetidine, and catecholamine excitation was selectively blocked by propranolol (1×10^{-5} M). The results show that human gastric mucosa contains at least two separate hormone-sensitive adenylate cyclases coupled to different receptors: one being responsive to histamine and the other to catecholamines. The demonstration of a histamine-sensitive adenylate cyclase system in human gastric mucosa is consistent with the concept that cAMP is the intracellular mediator of the secretagogue effect of histamine in man as well as in amphibia and rodents.

8022 CHLORIDE TRANSPORT AND ACID SECRETION IN STOMACH. (Eng.) Durbin, R. P. (Cardiovascular Res. Inst., Univ. California San Francisco, San Francisco, CA 94143). *Gastroenterology* 73(4, Part 2):927-930; 1977.

Different approaches to identifying the cellular Cl^- pool to facilitate the localization of the several modes of Cl^- transport in isolated frog gastric mucosa are illustrated. Flux studies on the effects of stimulating acid secretion and of removing secretory Cl^- on movement of Cl^- from nutrient to secretory solution and on the effects of removing nutrient Cl^- on the movement of Cl^- from secretory to nutrient solution showed that only a few minutes were required for the isotopic flux to reach a steady state. Cellular Cl^- uptake was quantitated by determining the extracellular space, using ^3H-mannitol. The overall mannitol space of 54% did not differ significantly from that reported for the mannitol space in tied mucosal sacs and was fairly close to the inulin space of 47-52% quoted for mounted mucosae. Therefore, the mannitol space seems to be a reasonable estimate of the extracellular space. The large mannitol space of 47% labeled from the nutrient solution corresponded to the serosal layer of connective tissue prominent in histological sections. The total tissue Cl^- (56.1 mEq/kg) was close to the values for freshly dissected mucosae (51.6 mEq/kg) and for incubated mucosae (68.0 mEq/kg) that were previously reported by one group of investigators; smaller levels found in other studies may reflect an incomplete extraction of Cl^- from the tissue. Further studies showed that over one-half of the cell Cl^- was taken up into the mucosal cells in 2 min. This rate of Cl^- uptake was compared to acid production using the average wet weight per unit area in these experiments of 53 mg/cm^2; cell Cl^- uptake was calculated to be 4.9 $\mu Eq/cm^2/hr$, a value slightly larger than the rate of acid secretion. The results suggest that much of Cl^- uptake occurs by exchange diffusion of Cl^- at the secretory surface.

8023 INTERACTIONS OF GASTRIC BLOOD FLOW, BARRIER BREAKER, AND HYDROGEN ION BACK DIFFUSION DURING ULCER FORMATION IN THE RAT. (Eng.)

Mersereau, W. A.; Hinchey, E. J. (Montreal General Hosp., Montreal, Quebec H3G 1A4, Canada). *Surgery* 83(3):248-251; 1978.

The interactions among gastric blood flow, H^+ back-diffusion, and the effects of barrier breakers were studied in a rat gastric chamber preparation to clarify the respective roles of these parameters in the pathogenesis of acute gastric erosions. Application of sodium taurocholate (STC) to the entire mucosal surface markedly increased H^+ loss from the chamber in the normal flow study (mean, 133.1 ± 6.5 μEq/15 min), whereas reduction of the area of barrier break by 50% reduced the H^+ loss proportionally (mean 61.7 ± 7.2 μEq/15 min). The application of STC to an area of 1 cm^2 (15% barrier break) led to a H^+ loss of 23.2 ± 4.4 μEq/15 min, which was not significantly different from the control period. No mucosal lesions were found where mucosal blood flow was present. Interruption of blood flow markedly reduced the rate of H^+ back-diffusion, as compared with normal flow groups in which either 100% or 50% of the area was covered with STC, but there was no significant difference with 15% barrier break whether blood flow was interrupted or not. Complete ischemia resulted in ulceration, which was limited to the area pretreated with STC. The application of STC to the squamous epithelium-lined forestomach failed to increase the permeability of this membrane to H^+ or Na^+ or to increase fluid flux. Sodium entry into the chamber was noted after barrier break in the normal flow group; however, the rate was not directly proportional to the area of barrier break and was less as the area of barrier break decreased. Under conditions of complete ischemia, sodium influx was markedly reduced. It is concluded that, although H^+ back-diffusion is a convenient measure of barrier permeability under conditions of normal mucosal blood flow, during mucosal ischemia this parameter fails to reflect permeability because of decreased mucosal clearance of H^+. These findings would explain apparent controversy whereby ulceration occurs under shock conditions in the absence of apparent H^+ back-diffusion.

8024 EVIDENCE FOR H_2-RECEPTOR-MEDIATED FEEDBACK REGULATION OF HISTAMINE RELEASE FROM ENDOCRINE CELLS IN THE RAT STOMACH. (Eng.) Hakanson, R.; Larsson, L. I.; Liedberg, G.; Sundler, F. (Dept. Pharmacology, Univ. Lund, Lund, Sweden). *J. Physiol. (Lond.)* 276:151-157; 1978.

The effects of histamine H_2-receptor agonists and of the blockade of H_2-receptors on pentagastrin-induced reduction of gastric histamine release was studied in male Wistar or Sprague-Dawley rats. The gastric mucosal histamine level was measured after s.c. injection of histamine dihydrochloride (0, 5, 10, or 25 mg/kg) or infusion of histamine (50 mg/kg/hr) or dimaprit (100 mg/kg/hr) with pentagastrin injections of 1, 62.5, 125, 250, or 500 μg/kg. Blockade by the H_2-receptor antagonists, metiamide (30 mg/kg/hr or 10 mg/kg) and burimamide (20 mg/kg), and by the H_1-receptor antagonist, mepyramine (30 mg/kg) was studied. In doses above 62.5 μg/kg, pentagastrin produced a 20-30% reduction of gastric histamine level 1 hr after injection ($p<0.001$ compared with untreated controls). Injected histamine inhibited this reduction if the histamine dose was high and the pentagastrin dose was low. Infused histamine or dimaprit prevented reduction of gastric histamine level at all dose levels of pentagastrin. When infusion of these receptor agonists was accompanied by infusion of the H_2-receptor antagonist, metiamide, the pentagastrin-induced release of histamine declined significantly ($p<0.001$) relative to saline-treated controls. The reduction of gastric mucosal histamine level after treatment with burimamide and pentagastrin was significant ($p<0.005$), as was the reduction caused by treatment with metiamide and pentagastrin ($p<0.001$) relative to rats treated with saline and pentagastrin. No significant lowering of gastric histamine content was noted when mepyramide and pentagastrin treatment was used. Similar results were obtained in intact and in antrectomized rats. To produce similar effects in vagotomized rats, higher doses of pentagastrin were required. The present study demonstrates a direct effect of the H_2-receptor antagonists on the histamine-storing endocrine-like cells in the oxyntic mucosa in the rat stomach. The effect is described as H_2-receptor mediated autofeedback, a mechanism whereby mobilized histamine inhibits further release of histamine from the cells.

8025 INHIBITION OF HISTAMINE-N-METHYLTRANSFERASE AND HISTAMINASE (DIAMINE OXIDASE) BY A NEW HISTAMINE H_2-RECEPTOR AGONIST, DIMAPRIT. (Eng.) Shaff, R. E.; Beaven, M. A. (Natl. Heart, Lung, Blood Inst., Natl. Inst. Health, Bethesda, MD 20014). *Biochem. Pharmacol.* 26(21):2075-2078; 1977.

The effects of Dimaprit on the *in vitro* activities of histamine decarboxylase (HD), diamine oxidase (DAO), and histamine-*N*-methyltransferase (HNMT) were investigated. HD was prepared from rat gastric mucosa, DAO from rat ileum, and HNMT from frozen guinea pig brain. Dimaprit was tested at 10^{-6}, 10^{-5}, 10^{-4}, 10^{-3}, and 2.5×10^{-3} M. Dimaprit had no effect on HD; it inhibited both DAO and HNMT noncompetitively, with apparent K_I values of $7\text{-}9 \times 10^{-6}$ M for HNMT and $2.5\text{-}3.0 \times 10^{-4}$ M for DAO. All values were the average of duplicate experiments. The results indicate that Dimaprit is a weak inhibitor of DAO and a stronger inhibitor of HNMT.

8026 IS HISTAMINE A MEDIATOR IN BILE-INDUCED GASTRIC MUCOSAL INJURY? (Eng.) Cheung, L. Y.; Porterfield, G. (Veterans Admin. Hosp., St. Louis, MO 63110). *J. Surg. Res.* 24(4):272-276; 1978.

The protective effect of histamine$_1$- and histamine$_2$-receptor antagonists on the mucosal permeability barrier and on gross mucosal injury was

evaluated in anesthetized dogs. After i.m. injection of mepyramine (10 mg/kg) and continuous i.v. infusion of cimetidine (50 μg/kg/min), measurements of ion fluxes and the transmucosal potential difference (PD) were made, and the degree of mucosal injury was scored by an independent observer. No evidence of mucosal injury was seen in any of the dogs before the introduction of 5.0 mM Na taurocholate. Measurements continued after the taurocholate was added to the HCl bathing solution of the test mucosa. The degree of mucosal injury in antihistamine-treated dogs was not significantly different from that in untreated dogs (lesion index, 1.9 ± 0.3 versus 1.6 ± 0.5, resp.). The PD in control mucosa did not change over the course of the experiment. Antihistamine treatment did not change the PD; however, exposure of the test mucosa to taurocholate resulted in an immediate drop in PD ($p<0.01$). The net flux of H^+ was not altered by the administration of the H_1- and H_2-receptor antagonists. Following exposure of the test mucosa to taurocholate, there was a marked increase in H^+ loss ($p<0.05$) compared with control mucosa or test mucosa before the addition of taurocholate. The change in Na^+ efflux was similar to that of H^+. Exposure to taurocholate resulted in a significant ($p<0.05$) increase in sodium efflux into the lumen. Comparison of H^+ flux in antihistamine-treated dogs and in untreated controls showed that exposure to taurocholate in either group resulted in a significant increase in luminal loss of H^+ and that the rate of H^+ back-diffusion increased slightly in the treated dogs. The failure of antihistamines to prevent bile salt-induced disruption of the permeability barrier and to protect against gross mucosal injury does not support the concept that histamine is a mediator in bile salt injury.

8027 PROPERTIES OF K^+/H^+ EXCHANGE ATPase FROM DOG GASTRIC MUCOSA. (Eng.) Scholes, P.; Lee, J. (Imperial Chemical Industries, Ltd., Mereside, Alderley Park, Macclesfield, Cheshire SK10 4TG, England). *Acta Physiol. Scand.* (Suppl.):427-434; 1978.

The suggestion that K^+ entry into vesicles prepared from dog gastric mucosa is important in maintaining optimum activity of the electrically neutral ATP hydrolysis-driven transport of H^+ into these vesicles was tested, and the hypothesis that K^+ ions act as counterions for H^+ transport was investigated. H^+ transport into microsomal vesicles from dog gastric mucosa was completely abolished by nigericin (2.5 μg/ml), which catalyzes a tightly coupled exchange of K^+ and H^+. Since this abolition could not be achieved by valinomycin (1.25 mg/ml) or FCCP (4.5×10^{-6} M) alone but only in combination, it appears that nigericin catalyzes an exchange of one H^+ ion for one K^+ ion. Optimum proton transport driven by ATP proceeded only when K^+ ions were present within the vesicles. Thus, proton transport was greatly diminished in media in which the K^+ ions were replaced by Na^+ ions or choline ions. However, when K^+-loaded vesicles were prepared, proton transport proceeded rapidly, irrespective of the type of cation in the external medium. ATP hydrolysis-driven transport of H^+ ions was only moderately reduced when Cl^- ions were replaced by glucuronate or SO_4^{2-} ions, suggesting that Cl^- ions are not important in the exchange of H^+ and K^+ ions *per se*. However, the slow phase observed in the time-course of ΔpH in the absence of Cl^- was similar to that observed in low K^+ media. This finding suggests that the equilibration of K^+ during the 15 min preincubation prior to the addition of MgATP is decreased in the absence of Cl^- ions. The cotransport of K^+ and Cl^- ions across the luminal membrane of the parietal cell in parallel with the H^+/K^+ exchange ATPase might be an important mechanism for HCl secretion.

8028 EFFECT OF METABOLIC ACIDOSIS ON GASTRIC ACID SECRETION AND SERUM GASTRIN. (Eng.) Vogel, S. B.; Ishiguro, N.; Manabe, M.; Eisenberg, M. M. (Univ. Minnesota Medical Sch., Minneapolis, MN). *Surg. Forum* 28:370-372; 1977.

The role of metabolic acidosis in acid secretion and the effect of metabolic acidosis on direct vagal parietal cell response and vagal antral gastrin mechanisms of acid production was investigated. Gastric acid secretory and serum gastrin level responses were determined in six mongrel dogs with standard gastric fistulas during mild (pH 7.2-7.3; 2% NH_4Cl infusion, 3-4 hr) and severe (pH 7.1-7.2; 4% NH_4Cl infusion, 3-4 hr) metabolic acidosis alone, following maximal parietal cell (160 mg/kg/hr histamine) and vagal (150 mg/kg, i.v., 2-deoxy-D-glucose [2DG]) stimulation, and following superimposition of parietal cell or vagal stimulation on mild or severe acidosis. All experiments were repeated after the dogs underwent truncal vagotomy. Peak acid output (PAO) in mild and severe acidosis was 7.4 ± 2.9 and 5.1 ± 2.3 mEq H^+/30 min, resp., and occurred after 5-6 hr of NH_4Cl infusion. Truncal vagotomy produced a marked reduction of PAO in mild acidosis but only a 50% reduction in severe acidosis (0.5 versus 2.2 ± 1.1 mEq H^+/30 min, resp.). Both mild and severe acidosis markedly reduced histamine-stimulated gastric acid secretion (from 26.2 ± 1.5 to 19.7 ± 1.4 or 13.7 ± 1.5 mEq H^+/30 min, resp.). The 40% reduction in gastric acid secretion following truncal vagotomy was only slightly augmented by mild acidosis and was unaffected by severe acidosis. Only mild acidosis affected 2DG-stimulated acid secretion; PAO after 2DG was 10.2 ± 3.4 mEq H^+/30 min and rose to 18.6 ± 4.4 mEq H^+/30 min during mild acidosis plus 2DG. Truncal vagotomy abolished the effect of 2DG alone; but small yet important acid secretory responses persisted during mild and severe acidosis (1.4 ± 0.4 and 2.8 ± 1.2 mEq H^+/30 min, resp.). The serum gastrin level increased from a control value of 29 ± 3 pg/ml to 82 ± 17 pg/ml in mild acidosis and to (34 ± 14)-(92 ± 17) pg/ml (range) in severe acidosis; truncal vagotomy abolished these effects. 2DG stimulation increased the serum gastrin level (from 22 ± 6 to 49 ± 12 pg/ml); further increases occurred during mild (from 21 ± 4 to 60 ± 13 pg/ml) and severe acidosis (from 23 ± 4 to 96 ± 7 pg/ml) and were abolished by truncal

vagotomy. It is concluded that the effects of metabolic acidosis on gastric acid and serum gastrin responses depend largely on intact vagal pathways, that metabolic acidosis has a direct effect on parietal cells, and that NH_4Cl or acidosis either decreases the sensitivity of the parietal cell or interferes in the histamine-parietal cell axis.

8029 THE EFFECTS OF ASPIRIN AND OTHER NON-STEROID ANTI-INFLAMMATORY/ANALGESIC DRUGS ON GASTRO-INTESTINAL MUCUS GLYCOPROTEIN BIOSYNTHESIS IN VIVO: RELATIONSHIP TO ULCEROGENIC ACTIONS. (Eng.) Rainsford, K. D. (Univ. Tasmania Medical Sch., G.P.O. Box 252C, Hobart, Tasmania, 7001, Australia). *Biochem. Pharmacol.* 27(6):877-885; 1978.

The effects of aspirin and related nonsteroid anti-inflammatory (NSAI) drugs on the incorporation of radio-labeled precursors into mucus glycoproteins isolated from the rat gastrointestinal tract were studied. The p.o. administration of aspirin at 200 mg/kg caused a marked inhibition of the incorporation of $^{35}SO_4^{-2}$ into isolated gastric glycoproteins at 5 hr or later following drug administration. In contrast, glycoprotein synthesis inhibition was only evident 24 hr after the administration of the same dose of salicylic acid. Aspirin failed to inhibit the incorporation of L-[U-^{14}C]threonine or [1-^{14}C]acetate into proteins or glycoproteins, resp., when these labeled precursors were given i.p. 5 min after the drug. To consider the possibility that the diverse systemic effects of salicylates may alter the pattern of organ uptake or metabolism of labeled acetate, radioactive acetate was given p.o. 5 min after the aspirin dose. Aspirin inhibited the incorporation of radioactive acetate, but the total uptake of [1-^{14}C]acetate by the gastric mucosa tissue was also reduced under these conditions, suggesting that the apparent inhibitory effect could be due to variable effects of the drug on uptake of the radioactive acetate. Of the NSAI tested (i.e., diclofenac, flurbiprofen, indomethacin, suprofen, azopropazone, dextropropoxyphene napsylate, diflunisal, fenclofenac, flufenamic acid, naproxen, paracetamol, and sulindac), only indomethacin, diclofenac, flurbiprofen, and suprofen caused inhibition of $^{35}SO_4^{-2}$ incorporation into gastric mucosal glycoproteins. When correlated with the gastric ulcer indices of these compounds, it was evident that the more potent ulcerogenic drugs caused a statistically significant reduction in radiosulfate incorporation, whereas those with little or no ulcerogenic activity did not. All salicylates tested caused an inhibition of $^{35}SO_4^{-2}$ uptake into glycopeptides isolated from the gastric mucosa incubated *in vitro*, with salicylate being a more potent inhibitor than aspirin. Maximal gastric mucosal tissue concentrations of salicylates were achieved at 10-15 min after p.o. administration, which coincided with a peak of salicylates in the blood. Much lower concentrations of salicylates were present in the intestinal contents and mucosa compared with those in the stomach. There was a rapid and appreciable rate of hydrolysis of aspirin to salicylate in the gastric mucosa.

8030 EFFECT OF ELECTRIC VAGAL STIMULATION ON STRESS ULCER FORMATION IN RATS. (Eng.) Nagakawa, B.; Adler, R.; Fischer, C.; Matsumoto, T. (Hahnemann Medical Coll. & Hosp., 230 N. Broad St., Philadelphia, PA 19102). *Int. Surg.* 62(9):477-479; 1977.

8031 EFFECT OF SALBUMATOL ON ACUTE GASTRIC ULCERATION INDUCED BY INDOMETHACIN IN THE RAT. (Eng.) Fielding, L. P.; Raute, M.; Curwain, B. P. (St. Mary's Hosp. Medical Sch., Praed St., London W2 1NY, England). *Eur. Surg. Res.* 9(4):252-257; 1977.

8032 EFFECT OF PENTAGASTRIN ON GASTRIC MUCOSAL DNA AND RNA SYNTHESIS IN GUINEA-PIGS [Abstract]. (Eng.) Sewing, K. Fr.; Muller, D. (Dept. Pharmacology, Univ. Tubingen, Tubingen, W. Germany). *Acta Hepatogastroenterol. (Stuttg.)* 24(6):491; 1977.

8033 EFFECT OF ATROPINE ON ENDOGENOUS RELEASE OF SECRETIN AND ACTION OF SECRETIN ON PANCREATIC SECRETION [Abstract]. (Eng.) Kim, M. S.; Lee, K. Y.; Chey, W. Y. (Genesee Hosp., Rochester, NY 14607). *Fed. Proc.* 37(3):302; 1978.

8034 EFFECT OF SC-29333 ON GASTRIC MUCOSAL BLOOD FLOW AND SERUM GASTRIN IN THE DOG [Abstract]. (Eng.) Driskill, D. R.; Colton, D. G.; Phillips, E. L.; Poy, P.; Dajani, E. Z. (Searle Lab., Chicago, IL 60680). *Fed. Proc.* 37(3):373; 1978.

8035 THE VARIABLE EFFECTS OF METABOLIC ACIDOSIS ON H^+ ION BACK DIFFUSION IN THE CANINE STOMACH [Abstract]. (Eng.) Gurll, N. (Univ. Iowa Coll. Medicine, Iowa City, IA). *Clin. Res.* 26(2): 110A; 1978.

8036 ADENOSINE EFFECTS ON CHOLINERGIC NEUROTRANSMISSION IN THE RABBIT STOMACH IN VIVO [Abstract]. (Eng.) Gustafsson, L.; Hedqvist, P.; Fredholm, B. B.; Olundh, S. (Karolinska Inst., Stockholm, Sweden). *Acta Physiol. Scand.* 102(1): 69A-70A; 1978.

8037 A COMPARATIVE STUDY OF MUCUS FROM NORMAL, INTESTINALIZED GASTRIC MUCOSA AND FROM GASTRIC ASPIRATES OF PATIENTS WITH PERNICIOUS ANAEMIA. (Eng.) Schrager, J. (Royal Albert Edward Infirmary, Wigan, Lancashire, England). *Adv. Exp. Med. Biol.* 89:528-529; 1977.

8038 THE EFFECT OF BILE SALTS ON THE PHYSICAL PROPERTIES OF MUCUS. (Eng.) Marriott,

C.; Kellaway, I. W.; Martin, G. P. (Dept. Pharmacy, Univ. Nottingham, University Park, Nottingham, NG7 2RD, England). *Adv. Exp. Med. Biol.* 89:509; 1977.

8039 STUDIES ON SERUM ENZYME MARKERS FOR GASTRIC MUCOSAL DAMAGE [Abstract]. (Eng.) Barayamil, H.; Waldron-Edward, D.; Greenberg, L. (Div. Surgical Res., McGill Univ., Montreal, Quebec, Canada). *Clin. Res.* 25(5):687A; 1977.

8040 FAT DIGESTION IN THE STOMACH OF PREMATURE INFANTS [Abstract]. (Eng.) Hamosh, M.; Sivasubramanian, K. N.; Salzman-Mann, C.; Hamosh, P. (Dept. Physiology, Georgetown Univ., Washington, DC 20007). *Physiologist* 20(4):40; 1977.

8041 A METHOD OF REVERSIBLE PYLORIC LIGATION FOR THE STUDY OF ULCER PRODUCTION AND HEALING [Abstract]. (Eng.) Ellert, M. S.; Jones, R. D. (Southern Illinois Univ. Sch. Medicine, Carbondale, IL 62901). *Physiologist* 20(4):25; 1977.

8042 COMPARATIVE ULCEROGENICITY OF MESECLAZONE AND OTHER NONSTEROIDAL ANTI-INFLAMMATORY DRUGS (NSAID) FOLLOWING ACUTE AND REPEATED ORAL ADMINISTRATION TO RATS [Abstract]. (Eng.) Diamantis, W.; Melton, J.; Sofia, R. D. (Wallace Lab., Cranbury, NJ 08512). *Fed. Proc.* 37(3):605; 1978.

8043 EFFECT OF VITAMIN C AND ASPIRIN ON ACUTE GASTRIC LESIONS IN THE RAT [Abstract]. (Eng.) Konishi, F.; Lo, C. (Southern Illinois Univ., Carbondale, IL 62901). *Fed. Proc.* 37(3): 359; 1978.

8044 A LIMBIC NEUROGENIC MODEL FOR RAPID GASTRIC MUCOSAL ULCERATION [Abstract]. (Eng.) Innes, D. L.; Viola, L. R.; Tansy, M. F. (Temple Univ., Philadelphia, PA 19140). *Fed. Proc.* 37(3):303; 1978.

8045 BIOCHEMICAL CHARACTERIZATION OF DUODENAL ULCER: EFFECT OF PROPIONITRILE OR CYSTEAMINE ON TISSUE CATECHOLAMINES AND SEROTONIN LEVELS [Abstract]. (Eng.) Szabo, S.; Horner, H. C. (Peter Bent Brigham Hosp., Boston, MA 02115). *Fed. Proc.* 37(3):302; 1978.

8046 PHARMACOLOGY OF CP-26,154, A NOVEL ANTI-ULCER AGENT [Abstract]. (Eng.) Pereira, J. N.; Mears, G. A.; Leader, J. P.; Hohnke, L. A. (Central Res. Div., Pfizer, Inc., Groton, CT 06340). *Fed. Proc.* 37(3):302; 1978.

8047 RELATIONSHIP OF ENKEPHALIN AND ENDORPHIN IMMUNOREACTIVITY WITH D-CELLS AND G-CELLS OF THE STOMACH [Abstract]. (Eng.) Forssmann, W. G.; Helmstaedter, V.; Feurle, G. (Anatomical Inst. III, Univ. Heidelberg, Heidelberg, W. Germany). *Acta Hepatogastroenterol. (Stuttg.)* 24(6):488; 1977.

8048 PREVENTION OF ENDOTOXIN-INDUCED GASTRIC DAMAGE BY LIDOCAINE [Abstract]. (Eng.) Greenberg, L.; Waldron-Edward, D.; Weksberg, A. (Div. Surgical Res., McGill Univ., Montreal, Quebec, Canada). *Clin. Res.* 25(5):687A; 1977.

8049 DENERVATION HYPERSENSITIVITY OF THE GASTRIC PARIETAL CELLS AFTER A HIGHLY SELECTIVE VAGOTOMY [Abstract]. (Eng.) Ferrie, M.; Joffe, S. N. (Royal Infirmary, Glasgow G4 OSF, England). *J. Physiol. (Lond.)* 273(2):72P-73P; 1977.

8050 GLUCAGON AND SECRETIN IN ASPIRIN-INDUCED EROSIVE GASTRITIS [Letter to Editor]. (Eng.) Carmichael, H. A.; Nelson, L.; Russell, R. I. (Royal Infirmary, Glasgow, Scotland). *Br. Med. J.* 2(6102):1604; 1977.

8051 MUCUS CHANGES IN SOME LABORATORY ANIMAL MODELS OF GASTRIC DAMAGE. (Eng.) Green, A. P.; Turner, D. H. (Beecham Pharmaceuticals Res. Div., Pinnacles, Colharbour Rd., Harlow, Essex, England). *Adv. Exp. Med. Biol.* 89:508; 1977.

8052 PREVENTION BY MILD IRRITANTS OF GASTRIC NECROSIS PRODUCED IN RATS BY Na-TAUROCHOLATE [Abstract]. (Eng.) Chaudhury, T. K.; Robert, A. (Upjohn Co., Kalamazoo, MI 49001). *Fed. Proc.* 37(3):303; 1978.

8053 EFFECT OF OSMOLALITY ON GASTRIC ACID SECRETION AND GASTRIC EMPTYING IN MAN. (Eng.) Swierczek, J. S.; Teichmann, R. K.; Rayford, P. L.; Thompson, J. C. (Univ. Texas Medical Branch, Galveston, TX). *Surg. Forum* 28:349-350; 1977.

See also, 7974, 7981, 8110, 8132, 8133, 8134, 8165, 8232, 8253, 8260, 8266, 8270, 8271, 8272.

8054 PANCREATIC SECRETION OBTAINED BY ENDOSCOPIC CANNULATION OF THE MAIN PANCREATIC DUCT AND SECRETIN RELEASE AFTER DUODENAL ACIDIFICATION IN MAN. (Eng.) Hanssen, L. E.; Osnes, M.; Myren, J. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 13(3):325-330; 1978.

The effect of acid in the duodenum on pancreatic secretion of water, bicarbonate, and α-amylase and on the release of secretin was studied in 12 patients suffering from gastrointestinal disease but having normal endoscopic retrograde pancreatographies. The main pancreatic duct was cannulated with a teflon catheter by means of a side-viewing duodenoscope. Six of the patients received a duodenal infusion of 40 ml of 100 mM HCl over 5 min; the others served as controls for basal pancreatic secretion. Pancreatic juice was collected for 20-30 min in 5-min samples, and blood was drawn at intervals for immunoreactive secretin (IRS) determination by radioimmunoassay. In the control group, no significant change was observed in the IRS level (range, 1.4 ± 0.3 to 1.9 ± 0.4 pM), bicarbonate concentration (range, 72 ± 5 to 73 ± 7 mM), bicarbonate output (range, 0.04 ± 0.02 to 0.06 ± 0.03 mmol/5 min), α-amylase concentration (range, 464 ± 38 to 508 ± 36 kU/5 min), or the α-amylase output (range, 0.3 ± 0.1 to 0.4 ± 0.2 kU/5 min). Duodenal acidification produced an increase in the immunoreactive secretin level (IRS, peak at 7 min of 15.8 ± 2.2 pM) and in the pancreatic secretion of water and bicarbonate. In the third 5-min sample, the maxima for pancreatic flow rate (11.5 ± 2.7 ml/5 min, $p<0.02$), bicarbonate concentration (122 ± 6 mM, $p<0.02$), and bicarbonate output (1.4 ± 0.4 mmol/5 min, $p<0.02$) were reached. After acidification, the output of α-amylase was significantly ($p<0.02$) higher and the concentration of α-amylase decreased, reaching its lowest point in the third 5-min sample (137 ± 22 kU/l, $p<0.02$). Using the improved technique of endoscopic cannulation of the main pancreatic duct, the basal levels of pancreatic secretion and those stimulated by HCl were determined. The observed rise in plasma IRS levels is thought to be responsible for the increased pancreatic flow rate and bicarbonate output.

8055 THE EFFECT OF PREDOMINANTLY α-ADRENERGIC DRUGS, INDIRECT SYMPATHOMIMETICS AND DOPAMINE ON EXOCRINE PANCREATIC FUNCTION: STUDIES IN THE ISOLATED CAT PANCREAS. (Ger.) Heidbreder, E.; Sieber, P.; Heidland, A. (Medizinische Universitatsklinik, Josef-Schneider-Strasse 2, D-8700 Wurzburg, W. Germany). *Res. Exp. Med. (Berl.)* 172(1): 97-108; 1978.

The influence of predominantly α-adrenergic substances (norepinephrine, epinephrine [each 0.005-0.6 μmol, perfusion], phenylephrine [0.3-0.6 μmol, perfusion]), indirect sympathomimetics (ephedrine, tyramine [each 0.3-0.6 μmol, perfusion]), and dopamine (0.3-0.6 μmol; perfusion) on the exocrine function of the isolated pancreas of adult cats was studied. The injection of epinephrine and norepinephrine induced a rapid depression of flow rate and a simultaneous increase of the perfusion pressure. Phenylephrine, ephedrine, tyramine, and dopamine did not change the perfusion pressure or the hydrokinetic function (flow rate, calcium, and chloride secretion). The secretion of protein and enzymes was enhanced both in intact animals and in cats pretreated with reserpine (1-1.5 mg/kg, i.p.) or 6-hydroxydopamine (60 mg/kg, i.v.). The pancreatic protein secretion was inhibited by both α-adrenolytic and β-adrenolytic substances, as well as by tetracain (5×10^{-4} M, perfusion) and atropine (2.5×10^{-9}-2.5×10^{-7} M, perfusion). It is concluded that predominantly α-adrenergic substances stimulate pancreatic enzyme secretion.

8056 THE ACTION OF DOPAMINE ON BASAL PANCREATIC SECRETION IN MAN. (Ger.) Lankisch, P. G.; Koop, H. (Medizinische Universitatsklinik, Robert-Koch-Str. 40, 3400 Gottingen, W. Germany). *Dtsch. Med. Wochenschr.* 103(9):391-392; 1978.

The effect of dopamine (4 μg/kg/min, 45-min infusion) on basal pancreatic secretion was studied in five male volunteers. Dopamine failed to influence basal bicarbonate and enzyme (trypsin and amylase) secretion. This finding is contrary to observations in dogs, and indicates that dopamine may be infused for circulatory shock in acute pancreatitis without reservations.

8057 DIFFERENTIAL RATES OF DIGESTIVE ENZYME TRANSPORT IN THE PRESENCE OF CHOLECYSTOKININ-PANCREOZYMIN. (Eng.) Rothman, S. S.; Wilking, H. (Dept. Physiology, Univ. California, San Francisco, CA 94143). *J. Biol. Chem.* 253(10):3543-3549; 1978.

Previous studies in which a peptide mixture containing cholecystokinin-pancreozymin (CCK-PZ) had stimulated digestive enzyme secretion by the pancreas in a nonparallel manner were repeated using the original preparation, the pure CCK-PZ peptide, and a synthetic, biologically active, COOH-terminal octapeptide fragment of the whole molecule. When CCK-PZ was added to the medium bathing rabbit pancreas *in vitro*, digestive enzyme secretion was enhanced in a dramatic nonparallel fashion. Protein, trypsinogen, and chymotrypsinogen secretion all increased, but increases in the secretion of the two enzymes were strikingly dissimilar. For the peak period, trypsinogen secretion was increased 15 times the control rates, while chymotrypsinogen secretion was increased only about 4 times. This difference was maintained over the 3-hr course of the experiment and was seen over a considerable range of protein concentrations. The effect was stimulus-bound and could not be ascribed to any error in measurement. The response was essentially the same whether the pure CCK-PZ polypeptide or an impure mixture of peptides containing 25% CCK-PZ by weight was tested. Unlike CCK-PZ, acetyl-β-methylcholine (MCh) increased trypsinogen and chymotrypsinogen secretion in a roughly parallel or quantitatively equivalent

fashion. Chymotrypsinogen output averaged 25 times the control rates at the peak, with increases as large as 125-fold, over 6 times the average peak response to CCK-PZ. Similarly, the COOH-terminal octapeptide fragment of CCK-PZ produced parallel stimulation of the digestive enzymes; the chymotrypsinogen output average 14 times the control rate for the peak period and increases as great as 100-fold were observed. Samples of CCK-PZ-stimulated secretion did not inhibit either chymotrypsinogen activation or chymotrypsin activity, and thus, the apparent paucity of chymotrypsinogen in CCK-PZ-stimulated secretion cannot be attributed to the secretion of an inhibitor of activity or activation in response to CCK-PZ. Since the digestive enzymes are apparently stored as mixed arrays within individual secretory granules, the differential secretion observed cannot be explained by the exocytosis of their contents.

8058 ACTION OF FATTY ACIDS ON THE EXOCRINE PANCREATIC SECRETION OF THE CONSCIOUS RAT: FURTHER EVIDENCE FOR A PROTEIN PANCREATIC INHIBITORY FACTOR. (Eng.) Demol, P.; Sarles, H. (Institut National de la Sante et de la recherche medicale, 46 Chemin de la Gaye, Marseille 13009, France). *J. Physiol. (Lond.)* 275:27-37; 1978.

The inhibitory effect of the intraduodenal infusion of oleic acid on pancreatic protein secretion, observed in conscious bile duct-ligated rats, was investigated by determining which nutrients (i.e., pure oleic acid, sodium oleate dispersed in polysorbate 80, esterified oleic acid in the form of olive oil, caprylic acid, butyric acid, glucose, and NaCl) could induce this inhibition after the normal bile flow was reintroduced into the duodenum. In the first series of experiments, bile but not pancreatic juice was reintroduced. Pancreatic flow rate increased significantly during 60 min after starting an infusion of oleic acid, with a peak at 30 min (+37% of plateau); protein concentration increased slightly (+3.9%) at 30 min and then decreased rapidly and remained significantly decreased in comparison with basal values (-65% in the last experimental period). Sodium oleate dispersed in polysorbate 80 produced an increase in volume and decrease in protein similar to those seen with pure oleic acid. The volume increase with sodium caprylate was less than that with oleic acid; the initial increase in protein concentration and output was greater than with oleic acid but the delayed inhibition was less marked. Volume, protein concentration, and protein output increased slightly but not significantly during sodium butyrate infusion, and there was no delayed inhibition. When olive oil was infused, volume increased slowly and progressively and was significantly increased only during the last two periods. When both bile and pancreatic juice were reintroduced into the duodenum, the basal protein secretion was lower than in the first series. However, the decrease in protein concentration and output following the administration of oleic acid showed the same pattern as when pancreatic juice flow alone was diverted. The increase in volume produced by olive oil was delayed compared with the response to oleic acid but reached a slightly higher peak; pancreatic protein output was comparable to that seen after oleic acid infusion, and the delayed inhibition was even more marked (-70%). No modification in volume or protein secretion was seen during or after 5% glucose or 0.9% NaCl infusion. A 30% glucose infusion produced a delayed increase in volume, which was maximal 60 min after the end of infusion. Protein concentration remained stable. A 5.4% saline infusion induced a 32% decrease in protein concentration but did not modify the volume. Delayed secretion produced by intraduodenal oleic acid infusion is not dependent on bile or pancreatic juice. The action of oleic acid is not a pathological phenomenon. Fatty acids must be free or saponified but not esterified in the form of triglycerides to produce the response.

8059 PANCREATIC RESPONSE AND PLASMA SECRETIN CONCENTRATION DURING INFUSION OF LOW DOSE SECRETIN IN MAN. (Eng.) Schaffalitzky de Muckadell, O. B.; Fahrenkrug, J.; Watt-Boolsen, S.; Worning, H. (Bispebjerg Hosp., Bispebjerg Bakke 23, DK-2400 Copenhagen NV, Denmark). *Scand. J. Gastroenterol.* 13(3):305-311; 1978.

To elucidate the physiological role of secretin in the regulation of pancreatic secretion, the secretin concentration in peripheral venous blood and the bicarbonate secretion into the duodenum was measured in 10 normal human subjects during i.v. infusions of low doses of secretin. The duodenum was intubated and fasting secretion of bicarbonate was measured during gastric aspiration. Afterwards, pancreatic secretion was stimulated by secretin at dose levels of 0.01, 0.03, 0.1, and 0.3 clinical U (CU)/kg/hr. The concentration of secretin in infusates (actual stimulation attained, 85%) and in venous plasma was measured by radioimmunoassay. The obtained increase in plasma secretin concentration (actual concentration minus fasting concentration) showed a linear correlation (r=0.915) with the measured secretin infusion rate. The median metabolic clearance rate was 15.7 ml/kg/min and the median half-life of exogenous secretin was 3.8 min. Physiological plasma concentrations of human secretin (below 10 pM) were obtained at doses of secretin lower than 0.1 CU/kg/hr. The corresponding levels of bicarbonate secretion ranged from 0 to 22.93 mmol/hr. The stimulated increase in bicarbonate concentration was linearly correlated (r=0.828) with the logarithm of the increase in venous concentration of secretin. The secretion of bicarbonate into the duodenum was markedly higher than in the fasting state even at the lowest rate of stimulation (the median concentrations were 0.41 pmol/hr during fasting, 1.35 pmol/hr at the 0.01-CU secretin dose, and 21.89 pmol/hr at the 0.30-CU secretin dose). The experiments show that even low doses of secretin can stimulate bicarbonate secretion in the duodenum. The secretin assay can detect a significant increase in secretin concentrations during infusion of a dose as low as 0.01 CU/kg/hr.

8060 INHIBITION OF SECRETIN-STIMULATED EXOCRINE PANCREATIC SECRETION BY DIAZOXIDE IN THE RAT. (Eng.) Pedrazzoli, S.; Dodi, G.;

Militello, C.; Favretti, F.; Brandstetter, S.; Lise, M. (Clinica Chirurgica Universita degli Studi, I-35100 Padova, Italy). *Eur. Surg. Res.* 10(1):33-39; 1978.

The effects of diazoxide, a known insulin inhibitor, on the basal, secretin-, and cholecystokinin-pancreozymin (CCK-PZ)-stimulated pancreatic exocrine secretion were studied in unanesthetized male Italicus rats. The rats were stimulated with an intra-aortic injection of secretin (18 U/kg), CCK-PZ (25 U/kg), or saline (1 ml, control). Doses of diazoxide (22.5 or 45 mg/kg) or saline (1 ml) were injected intra-arterially, 5 min before and 5 min after the stimulation. The volume of pancreatic juice and the protein and bicarbonate concentrations and outputs were determined at 10-min intervals. The basal pancreatic secretion was not modified by any of the doses of diazoxide used. Secretin significantly ($p<0.001$) increased the volume of pancreatic juice (from 5.16 ± 0.92 to 9.16 ± 1.72 µl/min) and the bicarbonate concentration (from 30.35 ± 3.9 to 47.49 ± 5.1 mEq/l) and output (from 156.60 ± 3.58 to 416.04 ± 6.70 mEq/min); these increases were inhibited by the higher dose of diazoxide while the lower dose reduced the volume response only. No significant variation in the pancreatic flow and bicarbonate concentration and output was seen in controls injected with diazoxide and saline or with saline only. Protein concentration and output were not significantly modified in rats treated with secretin or diazoxide or both. CCK-PZ significantly increased the protein concentration (from 2.47 ± 0.6 to 7.50 ± 0.95 g%, $p<0.001$) and protein output (from 77.73 ± 33.79 to 245.70 ± 38.90 µg/min, $p<0.01$). Diazoxide did not significantly influence these responses. Diazoxide did not modify the CCK-PZ-stimulated pancreatic secretion. The authors propose that the inhibition of insulin secretion by diazoxide may influence the interaction between secretin and the secretin receptors of the centroacinar and ductal cells. Direct action of diazoxide on these cells was ruled out because diazoxide did not modify the basal secretion. Since diazoxide inhibits the pancreatic exocrine response to secretin but does not modify the CCK-PZ stimulated secretion, it is suggested that insulin may have a permissive role on the secretin-stimulated pancreatic secretion.

8061 AMYLASE AND LIPASE SECRETION INTO BILE. (Rus.) Korotko, G. F.; Kurzanov, A. N. (Dept. Physiology and Biophysics, State Medical Inst., Andizhan, USSR). *Fiziol. Zh.* 64(1):81-89; 1978.

8062 PANCREATIC ACINAR CELL METABOLISM AND FUNCTION. (Eng.) Webster, P. D., III; Black, O., Jr.; Mainz, D. L.; Singh, M. (Veterans Admin. Hosp., Augusta, GA 30904). *Gastroenterology* 73(6): 1434-1449; 1977.

8063 NEUROHORMONAL CONTROL OF PANCREATIC SECRETION: A REVIEW. (Eng.) Singh, M.; Webster, P. D., III (Veterans Admin. Hosp., Augusta, GA 30904). *Gastroenterology* 74(1):294-309; 1978.

8064 FREE FATTY ACIDS AND LIPOLYTIC ACTIVITY OF THE PANCREAS OF IRRADIATED RATS. (Eng.) Kocmierska-Grodzka, D. (Inst. Physiology and Biochemistry, Medical Acad., 15-222 Bialystok, Poland). *Pol. J. Pharmacol. Pharm.* 29(5):533-539; 1977.

See also, 7995, 8047, 8115, 8132, 8396.

SECRETION AND METABOLISM

Hepatobiliary Tract

8065 DETERMINATION OF THE BILIARY TREE CAPACITY OF THE RAT IN ITS DISTENDED AND NONDISTENDED STATE USING ^{35}S-SULFOBROMOPHTHALEIN GLUTATHIONE (^{35}S-BSP-GSH) AS A MARKER. (Eng.) Dammann, H. G. (I. Medical Clinic, Univ. Hamburg, 2 Hamburg 20, W. Germany). *J. Physiol. (Paris)* 73(1):37-46; 1977.

BSP-glutathione was used as a marker to measure the biliary tree volume of male Wistar rats. Two methods were used to calculate the volume in the distended and nondistended state; one method is a modification of a previously described method in which biliary dead space is calculated as the volume of bile collected between the i.v. injection of ^{35}S-BSP-glutathione and its appearance in collected bile, while the other is a new method based on the assumption that the first measurable radioactivity following marker injection indicates the time of appearance of the injected marker in the right and left hepatic duct. The biliary tree capacity in the nondistended state was only 77.8% of that reported in previous studies when determined by the first method and 72% of previous values when determined by the second method. The values were 30.4 ± 5.2 and 20.0 ± 3.7 µl by the first and second methods, resp., corresponding to 2.49 ± 0.37 and 1.67 ± 0.42 µl/g liver; the differences between the values obtained by the two methods were statistically significant in both cases ($p<0.05$). The distended biliary

tree volume was determined two different ways. In one set of experiments, ^{35}S-BSP-glutathione was injected i.v. 2-3 sec before terminating a 15-min biliary obstruction; in the other experiments, the injection was administered 60 sec before terminating the struction. The new method for calculating biliary volume was used in both cases. The distended volume calculated after injecting the marker 2-3 sec before terminating biliary obstruction was 69.8 ± 7.1 µl or 5.07 µl/g liver, whereas the values obtained after injecting the marker 60 sec before terminating the obstruction were 59.6 ± 6.7 µl and 4.24 ± 0.94 µl/g liver. These levels were all significantly lower ($p<0.05$) than those calculated using the previously published method (119.8 ± 12.7 µl and 8.84 µl/g liver). The capacity was also significantly lower when the marker was injected at 60 sec than at 2-3 sec. It is concluded that determining biliary tree volume by administering the marker 60 sec before terminating an obstruction gives a more accurate estimate of the true biliary tree capacity in the distended state.

8066 EXPERIMENTAL STUDIES ON CONTINUOUS HYPOTHERMIC LIVER PERFUSION WITH A SYNTHETIC SOLUTION CONTAINING GELATIN POLYPEPTIDES (HAEMACCEL). (Eng.) Fuller, B. J.; Attenburrow, V. D.; Newsome, C. (Royal Free Hosp. Sch. Medicine, London, England). *Cryobiology* 15(3):279-289; 1978.

A rat liver model was used to study preservation by continuous hypothermic perfusion, and experiments were designed to investigate whether changing the perfusion pressure would alleviate the problems of increased resistance to flow encountered during hypothermic liver perfusion. Perfusion for 5 or 24 hr with a defined solution containing Haemaccel as colloid at a temperature of 5-7 C was found to result in good preservation of the metabolic activity of the livers, as assessed by the ability of the tissue to synthesize urea (8.89 ± 1.34 µmol/g at 5 hr, 7.18 ± 1.23 µmol/g at 24 hr; both perfusions at 20 cm H_2O) and to take up galactose (40.78 ± 4.83 µmol/g at 5 hr, 34.76 ± 5.36 µmol/g at 24 hr). In common with other continuous perfusion methods, there was increasing evidence of cell damage as perfusion progressed; at 5 hr, biopsy specimens showed clearly visible sinusoidal channels and a well preserved chain-like formation of the hepatocytes, whereas at 24 hr, there were regions in which the sinusoidal spaces were small or indistinguishable, even though the adjacent branches of portal vein, artery, and central vein were patent. In addition, there was a depletion of cellular K^+ ($p<0.05$) and an increase in tissue water content ($p<0.01$) in 24-hr perfused livers compared with 5-hr perfused livers. There was a considerable increase in vascular resistance, as indicated by a fall in flow rate at a constant pressure, in the later stages of perfusion; flow rate after 24 hr was reduced to approximately 30% of that after 5 hr. Further studies are necessary to analyze, in a controlled fashion, the nature of the damage incurred by the liver during storage and the requirements for maintenance of a viable graft by hypothermia.

8067 CORRECTION OF HEPATOCELLULAR DYSFUNCTION DURING ENDOTOXEMIA. (Eng.) Heimberger, S. L.; McDougal, W. S.; Wilmore, D. W.; Pruitt, B. A., Jr. (United States Air Force Medical Center, Keesler Air Force Base, MS 39534). *J. Surg. Res.* 24 (5):442-448; 1978.

Because endotoxin exerts a variety of effects on physiologic function, a nonlethal endotoxemia model was established in dogs to examine its effect on the alterations in transport function of the hepatocyte. Indocyanine green (ICG) was used as a clearance marker of hepatic membrane transport, and the impact of energy substrate on hepatic transport was determined. No change in hepatic blood flow, plasma volume, or blood pH or gas concentrations was observed following the i.v. injection of *Escherichia coli* endotoxin, given as 0.1 mg/kg body weight of lipopolysaccharide followed by a 0.05-mg/kg/hr infusion for 2 hr. However, marked alterations in the kinetics of ICG occurred following administration; the initial rapid extraction reflected by the K_1 rate constant decreased ($p<0.001$) and back diffusion, thought to suggest increased membrane permeability (reflected in part by the K_2 constant), increased ($p<0.01$). These alterations in dye clearance suggest a direct effect of endotoxin on hepatocyte transport, substantiating the earlier observations of similar changes that occur in bacteremic, thermally injured patients. The alterations in hepatic energetics were returned to normal by the simultaneous administration of a 50% glucose solution (2 g/kg) and insulin (0.2 U/kg), followed by an infusion of 0.5 g glucose/kg/hr and 0.2 U insulin/kg/hr. In this model system, endotoxin is thought to have a direct effect on the hepatocyte, markedly altering cellular energetics and hepatocyte transport, and its action may account for the progressive changes in hepatic function that are frequently observed in critically ill infected patients.

8068 THE USE OF FINE-NEEDLE ASPIRATION BIOPSY FOR THE DIAGNOSIS OF HEPATIC STEATOSIS IN RATS. (Eng.) Boelsterli, U.; Zbinden, G. (Inst. Toxicology, Univ. Zurich, Schorenstrasse 16, 8603 Schwerzenbach, Switzerland). *Toxicol. Lett.* 2(1): 35-43; 1978.

The value of fine needle aspiration biopsy (FNAB) for the examination of experimental liver lesions in small laboratory animals was investigated in male ZUR:SIV-Z strain rats with experimental fatty liver. Rats were fed a high fat diet (39.7% butter, 5% cholesterol) with or without simultaneous treatment with chlortetracycline (80 mg/kg, intragastrically, 5 times/week), and FNAB was performed on days 2, 4, 8, and 16 of treatment. The specimens were stained with the Masson-Goldner and Van Gieson methods (MGG) or with hematoxylin and Fettrot 7B. The appearance of these biopsy specimens was compared with that of specimens obtained at necropsy and stained by the same methods. After MGG staining, the cytoplasm of the cells taken by FNAB from the untreated control rats had a fine granular appearance. Stainable fat was not increased in the

chlortetracycline-treated rats receiving a normal diet. In animals fed the high fat diet, with and without chlortetracycline, diffuse vacuolization was seen in the MGG-stained smears, and a distinct accumulation of fat droplets was seen in cells stained with Fettrot 7B and hematoxylin. This accumulation was seen even after 2 days, and the number of fat droplets increased over the study period. The degree of fatty change was scored using arbitrary units in both the specimens taken by biopsy and those taken at necropsy. Scoring was identical in both the biopsy and necropsy specimens in 60% of the cases. In 38%, scoring differed by 1 unit, and the higher score was usually obtained in the necropsy slide. The scores in the two specimens differed by 2 units in only two cases. The safety of FNAB was confirmed by repeated puncture of the same liver lobe (40 punctures in 10 weeks). This treatment resulted in small necroses and hemorrhages that were rapidly organized without significant distortion of the liver structure. It appears that FNAB is useful in detecting early morphologic liver changes and in monitoring their progression, without the necessity of sacrificing large numbers of animals.

8069 A COMPARISON OF METHODS FOR BILE SALT POOL SIZE MEASUREMENTS IN THE PRAIRIE DOG GALLSTONE MODEL. (Eng.) Meyer, P. D.; DenBesten, L. (Veterans Admin. Hosp., Iowa City, IA 52242). *Proc. Soc. Exp. Biol. Med.* 156(3):452-456; 1977.

A modified isotope-dilution technique for measuring bile salt pool size was compared with the wash-out technique in the prairie dog gallstone model. Cholelithiasis was induced by feeding high-cholesterol chow. Fasting animals received ^{14}C-labeled cholic acid i.v. and a gavage feeding of a lipid-protein emulsion to stimulate gallbladder contraction. Twelve hours later, the cystic duct was ligated, and the common duct was cannulated. Gallbladder bile salt and bile salt from the first period hepatic collection were determined by mass, and radioactivity was also determined. The radioactivity administered was corrected for losses by multiplying by e^{-kt}, where k is the fractional pool loss per unit time (t). The previously reported isotope-dilution technique (which mathematically assumes no losses, so that k=0 and e^{-kt}=1) was used to estimate pool size in computing k; the daily synthesis rate was assumed to be 24 times the hourly synthesis rate as determined after wash-out. The radioactivity from gallbladder bile and the first collection of hepatic bile was subtracted from this corrected radioactivity. This computation yields the radioactivity actually contained in the animal after the first hepatic bile collection. This radioactivity is divided by the specific activity of bile salt from the second-period hepatic bile collection; this constitutes the "pool remaining" in the animal after the first-period hepatic bile collection. The sum of the bile salt in the gallbladder, first-period hepatic bile, and pool remaining constitutes the total bile salt pool. The bile acid pool as calculated by the modified isotope-dilution technique was 333 ± 104 µmol in controls and was 276 ± 84 µmol as calculated by washout. The average basal synthesis rate was 2.51 ± 1.16 µmol/hr, determined as the "plateau" of the wash-out curve. The difference between the two methods varied in magnitude but was consistent and statistically significant ($p<0.001$). The pool size as measured by isotope dilution was 431 ± 137 µmol, and by wash-out was 357 ± 120 µmol ($p<0.001$). Basal synthesis rate was 2.45 ± 1.06 µmol/hr. Wash-out pools corrected for recovery of radioactivity agreed very well with pools calculated by isotope dilution. These studies indicate that the modified isotope-dilution technique yields values for bile salt pool sizes consistently higher than those of the wash-out technique. The observed differences in pool size measurement apparently reflect an underestimation by the wash-out technique, possibly due to some portion of the pool, previously equilibrated with exogenous isotope, being unavailable for "wash-out" and measurement.

8070 REGULATION OF THE VOLUME OF ISOLATED RAT HEPATOCYTES IN HYPOOSMOTIC MEDIUM. (Fre.) Poggioli, J.; Mazet, J. L.; Claret, M. (Laboratoire de Physiologie comparee et de Physiologie cellulaire, C.N.R.S. Universite Paris-XI, 91405 Orsay, Paris, France). *C. R. Acad. Sci. [D] (Paris)* 285: 1467-1470; 1977.

Changes in the volume of isolated rat hepatocytes were studied 2 min to 2 hr after incubation in minimum essential medium (MEM, 37 C, pH 7.4) with NaCl concentrations ranging from 0 to 256 mM. The effect of ouabain addition (10^{-3} M) to the MEM was also observed. MEM with 185 mM NaCl increased cell volume to 150% immediately after incubation; the cell volume decreased to 106% of the initial volume after 1 hr of incubation. MEM with 92 mM NaCl increased cell volume to 260% of the inital volume after 2 min of incubation; it decreased to 150% of the initial volume 2 hr after incubation. Ouabain had no effect on the initial increase in cell volume induced by NaCl; ouabain only slightly affected the long-term change in cell volume with MEM containing 185 mM NaCl and had no effect on the cell volume with MEM containing 92 mM NaCl. Potassium and amino acid losses from the cells were 82 nmol/mg and 60 nmol/mg, resp., after 150 min. Thus, potassium or amino acid loss from the hepatocytes incubated in MEM with NaCl had no effect on the regulation of hepatocyte volume. The authors suggest that other mechanisms, such as a sodium pump (as in the kidneys), a contractile membrane system, or a decrease in the activity of osmotic particles by their binding to intracellular sites, may be involved in the regulation of hepatocyte volume.

8071 DEMONSTRATION OF AUTOANTIBODIES IN LIVER DISEASE USING INDIRECT IMMUNOFLUORESCENCE. (Ger.) Storch, W. (Neurologische Klinik, Karl-Marx Universitat, Karl-Marx-Stadter-Strasse 50, DDR-7039

Leipzig, E. Germany). *Dtsch. Gesundheitsw.* 33(6): 257-265; 1978.

A synopsis of antibodies that appear in liver disease is presented. Antibodies are considered to be of diagnostic value and may provide information about pathogenesis, etiology, therapy, course of the disease, and prognosis. The antibodies, demonstrable by indirect immunofluorescence, include those against nuclear material (antinuclear factors), mitochondria, smooth muscle, microsomes, connective tissue, endothelium, bile ducts, bile duct epithelium, bile canaliculi, hepatic cytoplasm, immunoglobulins, thyreoglobulins, microsomal thyroid antigen, parietal cells, brush borders of proximal tubules in rat kidneys, lymphocytes and thrombocytes, collecting tubules, loop of Henle cells, and various proximal and distal renal tubule cells. Demonstration of humoral autoantibodies indicates that the tolerance to those particular antigens has been decreased. In liver disease, especially in chronic aggressive hepatitis, there are various groupings of disease, which may or may not be associated with antibodies. In differential diagnosis, specific antibodies demonstrated may be informative in cases of acute viral hepatitis, icterus of unknown etiology, chronic active hepatitis, lupoid hepatitis, and hepatitis of viral origin. Antibodies detected may provide clues about the identity of some forms of hepatitis responding to immunosuppressive therapy, about some forms possibly correlated with alpha-1-antitrypsin deficiency, and about the presence of active lupus erythematosus disseminatus.

8072 PREFERENTIAL LOCALIZATION OF RADIOLABELED LIPOSOMES IN LIVER. (Eng.) Hinkle, G. H.; Born, G. S.; Kessler, W. V.; Shaw, S. M. (Tulsa Medical Coll., Tulsa, OK 74114). *J. Pharm. Sci.* 67(6):795-798; 1978.

Liposome formulations were studied to achieve an efficient entrapment procedure for the production of liposomes of ^{99m}Tc-pentetic acid (^{99m}Tc-I). The entrapment efficiency was studied by separation of the product using column chromatography. The mean percentage of ^{99m}Tc-I activity in the liposome fractions of negatively charged liposomes, as calculated from a total of six entrapment studies, was 1.86 ± 0.34%. The mean percentage of ^{99m}Tc-I activity in the liposome fractions of positively charged liposomes, as calculated from 12 entrapment studies, was 8.14 ± 1.66%. A 60-min sonication time produced significantly lower ($p=0.05$) entrapment yields than did 15-, 30-, and 45-min times, which did not differ from each other significantly. The particle size range of the prepared liposomes was evaluated using electron microscopy. Studies of liposomes contained in the first fraction after the void volume in gel filtration showed the presence of liposomes ranging from 0.1 to 2 μm in diameter. Successive fractions contained small-diameter liposomes, which remained in the circulation for extended periods. Entrapment techniques and separation procedures led to a liposome preparation with particles in the colloidal size range (0.001-0.5 μm). Dramatic differences in the organ distribution of the liposome preparation in mice were produced when different particle size ranges were injected. Liposomes eluted in the first fraction after the void volume led to a maximum uptake by the liver and spleen 10 min after i.v. injection. Liposomes from pooled fractions provided less than half of the activity in the liver, as did the narrow size range liposome preparation. Investigations concerning improvement of the entrapment yield along with the production of a liposome product with an increased radioactive concentration are being planned. In addition, decreasing the labeling procedure time, which could lead to the development of a kit preparation suitable for human use, is being studied.

8073 RESOLVING BIOCHEMICALLY THE HEPATOCYTE'S MULTIFUNCTIONAL PERICELLULAR MEMBRANE. (Eng.) Evans, W. H. (Natl. Inst. Medical Res., Mill Hill, London, England). *Trends Biochem. Sci.* 2(3):169-171; 1977.

Studies concerning the hepatocyte pericellular membrane are reviewed. This membrane is complex and can be subfractionated to yield plasma membrane fractions from the blood sinusoidal, contiguous, and bile canalicular regions. The blood sinusoidal region is microvillar and accounts for 40-50% of surface area. Plasma membranes are vesicles of density 1.11-1.14 g/cm^3 in sucrose and are recovered mainly from microsomal fractions; this region is identified by perfusing the liver with ^{125}I-ligands. It has a high sialic acid and glycoprotein content. The contiguous region accounts for 30-40% of surface area; here the plasma membranes are of density 1.16-1.18 g/cm^3. Intercellular junctions are present. Biochemical properties include a low glycoprotein and glycolipid content and prominent gap-junctional polypeptides; this region has the lowest specific activities of plasma membrane marker ecto-enzymes. The canalicular region is microvillar and accounts for about 10% of surface area. These plasma membranes are isolated as canalicular complexes (density 1.16-1.18 g/cm^3) or vesicles (density 1.13 g/cm^3). This region has the highest specific activities of plasma membrane marker ecto-enzymes, cholesterol level, and sphingomyelin level. There is little activation of adenylate cyclase by glucagon, and there is a high sialic acid and glycoprotein content. Several studies have indicated that radioactivity first associated with macromolecules at the hepatocyte's Golgi apparatus is later found at the blood sinusoidal region of the pericellular membrane. Thus, it may be assumed that plasma membrane glycoproteins, after their insertion into the blood sinusoidal plasma membrane, can then redistribute laterally. Such a mechanism would explain the lower concentration of glycoproteins generally in plasma membranes derived from the lateral region of cells relative to membranes derived from microvillar and brush border areas. However, a major unresolved problem concerns the extent to which plasma membrane specializations constrain lateral redistribution of components.

8074 IDENTIFICATION OF ERYTHROPOIETIN PRODUCING CELLS IN FETAL MOUSE LIVER CULTURES. (Eng.) Gruber, D. F.; Zucali, J. R.; Miranda, E. A. (Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263). *Exp. Hematol.* 5(5):392-398; 1977.

Morphological studies were carried out to identify the fetal hepatic cell responsible for the production of erythropoietin in culture. Fetal mouse liver explants produced measureable amounts of erythropoietin in the culture media. Erythropoietin was first detected in pooled medium between days 2 and 7 of culture; the amount increased with time and was greatest between days 15 and 21. The addition of anti-erythropoietin to the 15-21 day culture medium neutralized the erythropoiesis-stimulating capability of the culture. Immunofluorescent and carbon particle ingestion techniques suggest that the erythropoietin is elaborated by macrophages or Kupffer cells of the hepatic reticuloendothelial system.

8075 THE BIOCHEMICAL PREREQUISITES FOR PREVENTING PATHOGENIC LYSOLECITHIN ACTIVITY IN THE HUMAN GALLBLADDER. II. STUDIES ON THE SUBCELLULAR LOCALIZATION OF LYSOPHOSPHOLIPASE. (Eng.) Tagesson, C.; Sjodahl, R. (Dept. Medical Microbiology, Univ. Linkoping, S-581 85 Linkoping, Sweden). *Scand. J. Gastroenterol.* 13(1):107-112; 1978.

The subcellular localization of lysophospholipase activity in the human gallbladder epithelium was studied by differential and density gradient centrifugation to determine whether the presence of this enzyme could protect the gallbladder wall against potentially pathogenic lysolecithin. Differential centrifugation revealed that lysophospholipase was enriched both in the lysosomal mitochondrial fraction (20,000 x *g* pellet) and in the microsomal fraction (100,000 x *g* pellet). The highest relative specific activity (2.27) was found in the microsomal fraction, whereas most of the enzyme activity (56%) was recovered from the lysosomal mitochondrial fraction. The soluble fraction (100,000 x *g* supernatant) contained only 6% of the total activity. Although the cytosol did not contain any significant lysophospholipase activity, large amounts of enzyme were solubilized during sucrose density gradient centrifugation. The findings suggest that a widespread subcellular distribution of lysophospholipase in the gallbladder epithelium serves a physiologically favorable purpose. The authors suggest that one purpose is to prevent the development of acute aseptic cholecystitis, but that this result is not achievable in cases in which the gallbladder epithelium is mechanically injured by an impacted stone.

8076 THE BILIARY EXCRETION OF BUCOLOME IN THE RAT: A POSSIBLE CAUSE FOR CHOLERESIS. (Eng.) Kitani, K.; Nokubo, M.; Kanai, S.; Miura, R.; Uesugi, T. (Tokyo Metropolitan Inst. Gerontology, 35-2 Sakaecho, Itabashiku, Tokyo 173, Japan). *Biochem. Pharmacol.* 26(24):2457-2461; 1977.

Because the mechanism of bucolome (BC)-induced choleresis has not been elucidated, the possibility that BC or its metabolite(s) might induce osmotic choleresis when excreted into the bile was examined by measuring the extent of such excretion in anesthetized 10-week-old male Wistar rats. Following i.p. administration of ^{14}C-BC mixed with carrier BC (20 mg/100 g body weight), the common bile duct was cannulated, and seven successive 15-min bile samples were collected beginning 15 min after BC injection. In a second group of rats cannulation occurred before BC administration, and 15-min samples were collected for 2 hr. In both groups, a previously-cannulated femoral artery provided arterial blood samples at various intervals. The highest average plasma BC level was 0.47 mg/ml in group I rats and 0.3 mg/ml in group II rats. The total biliary excretion of BC (a percentage of the administered dose) was 19.0 ± 4.0 in group I rats and 24.5 ± 4.0 in group II rats from 15 min to 2 hr of sampling. A significant (r=0.85) linear relationship was observed between bile flow rate and the bile excretion rate of BC, 27 µl of bile being produced for each µmole of BC excretion. These results indicate that in rats BC excretion in bile could be a major cause of choleresis, possibly due to the osmotic force of the excreted BC. It is possible that, in animals without a bile fistula, the enterohepatic circulation of BC could maintain a higher plasma BC level than that encountered in this study.

8077 METABOLISM OF THE CARCINOGEN CHROMATE BY RAT LIVER MICROSOMES. (Eng.) Gruber, J. E.; Jennette, K. W. (Dept. Chemistry, Dartmouth Coll., Hanover, NH 03755). *Biochem. Biophys. Res. Commun.* 82(2):700-706; 1978.

Studies were carried out to determine whether the oxidation state of chromium is affected in its metabolism by the rat liver microsomal systems, since the oxidation state of this element has been previously shown to be very important in determining the carcinogenicity and mutagenicity of chromium compounds and their effect on DNA synthesis. Incubation of chromate (4.0×10^{-4} M) with rat liver microsomes and 0.025 M NADPH resulted in reduction of hexavalent chromium to trivalent chromium. In the absence of NADPH, no reduction occurred. The amount of chromium reduced in the presence of NADPH depended on the concentration of the microsomes. With a microsomal protein concentration of 1.5 mg/ml, essentially complete loss of chromate was observed after 6 hr of incubation. Chromate reduction was also dependent on the concentration of NADPH. Maximum conversion to chromium III occurred at a NADPH concentration of 1.44×10^{-3} M upon incubation with 1.5 mg/ml microsomes for 6 hr. Time course studies at these concentrations showed that chromate was completely reduced to chromium III. Incubation of chromate with microsomes in the absence of NADPH led to complete recovery of chromate even after 24 hr in-

cubation, indicating that chromate did not oxidize any components of the microsomal system under these conditions. In the absence of microsomes, less than 20% of the chromate was reduced by NADPH, compared with a greater than 98% reduction with NADPH and microsomes. These results suggest that an NADPH-requiring enzyme or enzyme system is involved in the *in vitro* metabolism of chromate. The results of this study, together with those of previous studies, suggest that chromium III may be the ultimate carcinogenic form of chromium.

8078 EFFECT OF EXPERIMENTAL MALNUTRITION ON CORTICOSTEROIDS AND LIVER CYTOPLASMIC RECEPTORS IN RATS. (Eng.) Kadival, G. V.; Samuel, A. M. (Tata Memorial Hosp., Parel, Bombay 400 012, India). *Biochem. Med.* 18(3):353-359; 1977.

Adrenocortical function was studied in experimental malnutrition in male Wistar rats. Plasma corticosterone levels, liver cytosol binding capacity, and affinity binding constants were estimated in animals sacrificed on days 15, 21, and 28 after starting a low-protein (1% casein) diet. These parameters were also estimated in another group of animals that had been fed the low-protein diet for 4 weeks and were then given a normal diet; these animals were sacrificed on the 7th, 15th, and 21st days of repletion. Serum albumin levels declined markedly from control levels by the 15th day of the low-protein diet. Plasma corticosterone, liver cytosol binding capacity, and affinity constants for the cytosol binding protein remained unchanged during hypoalbuminemia and during replacement with the normal diet. Starvation was associated with hypertrophy of the adrenal glands, which was reflected by an increase in the weight of these glands. The weight returned to normal within 2 weeks on a normal diet. It is concluded that, even during progressive phases of malnutrition, the corticosteroid levels are maintained within normal limits, probably by a compensatory hypertrophy of the adrenal glands. The liver cytosol binding proteins are within normal limits and thus do not reduce the effectiveness of the biological action of corticosteroids.

8079 EFFECTS OF HEPATOTOXIC AGENTS ON HEPATIC MICROSOMAL METABOLISM OF ESTROGENS IN THE RAT. (Eng.) Lopez del Pino, V.; Bolt, H. M. (Institut fur Toxikologie der Universitat Tubingen, Wilhelmstr. 56, D-7400 Tubingen 1, W. Germany). *Arzeim. Forsch.* 27(11):2117-2120; 1977.

The effects of carbon tetrachloride- and ethanol-induced liver damage on the activities of the microsomal enzymes involved in estrogen metabolism were investigated in the livers of male Wistar rats. The animals were treated with ethanol, either acutely (6 ml/kg, p.o., and a 40% volume/volume aqueous solution) or chronically (20% volume/volume ethanol in drinking water for 100 days), or with CCl_4 (2.5 or 5 ml/kg of a 50% solution, p.o.); in further experiments rats received a single dose of thioacetamide (100-300 mg/kg). Acute or chronic ethanol pretreatment had no effect on the levels of liver microsomal cytochrome P-450, but acute and chronic treatment significantly reduced NADPH-cytochrome c reductase activity ($0.0125>p>0.01$) in comparison with control levels. Acute treatment decreased mestranol demethylation significantly ($0.01>p>0.005$), and chronic treatment decreased demethylation even further ($p<0.0005$). Acute or chronic ethanol treatment had no effect on the aromatic hydroxylation of estradiol, as indicated by microsomal replacement of tritium from 2,4,6,7-^{3}H-estradiol. More severe liver damage by CCl_4 or thioacetamide was associated with a reduction in microsomal cytochrome P-450 ($0.01>p>0.005$ and $0.0025>p>0.0005$ for 2.5 and 5.0 ml/kg CCl_4 doses, resp.; $0.05>p>0.025$ and $0.0025>p>0.01$ for various thioacetamide doses), whereas NADPH-cytochrome c reductase was not significantly diminished. Demethylation of mestranol was significantly inhibited by 2.5 and 5.0 ml/kg CCl_4 ($0.0125>p>0.01$ and $0.05>p>0.025$, resp.), as was aromatic hydroxylation of estradiol ($0.05>p>0.025$ and $0.0025>p>0.0005$, resp.). Although chronic thioacetamide treatment has been previously reported to decrease microsomal aromatic estrogen hydroxylation, a single dose (200 or 300 mg/kg) caused increased microsomal formation of estrone from estradiol ($0.0025>p>0.0005$ or $0.005>p>0.0025$, resp.), which is regarded to be a more lipophilic substrate for the microsomal estrogen 2-hydroxylase than is estradiol. These data show that hepatotoxic agents may act differentially on hepatic metabolism of endogenous steroids.

8080 PATHWAYS OF ETHANOL OXIDATION IN HEPATIC MICROSOMES OF ACATALASEMIC (Csb) MICE. (Eng.) Vatsis, K. P.; Schulman, M. P. (Univ. Illinois Medical Center, Chicago, IL 60612). *Adv. Exp. Med. Biol.* 85A:303-317; 1977.

A previous study suggesting that NADPH-dependent ethanol oxidation by hepatic microsomes (MEOS) is independent of catalase activity was reinvestigated using the same techniques in genetic control (Csa) and acatalasemic (Csb) mice. Csa and Csb microsomes kept in an ice bath at 0-4 C and subsequently incubated at 25 C or 30 C for 10 min showed identical MEOS and catalase activities, whereas incubation of similar preparations at 37 C for 10 min led to a 20-35% loss of catalase and a 10-30% decrease in MEOS activities of Csb as compared with corresponding activities in Csa mice. Based on these observations, MEOS assays were uniformly carried out at 30 C (10 min), regardless of microsomal origin or pretreatment. Preincubation of Csb microsomes at 42-44 C for 15 min resulted in 97-99% inactivation of catalase activity and a concomitant 60-70% loss of MEOS activity; neither the catalase nor MEOS activity of Csa microsomes was affected by this procedure. Preincubation at 43 C for 15 min had no effect on NADPH-linked drug-metabolizing activities of Csa or Csb microsomes. MEOS activity of "zero-time" microsomes from either strain, or of 43 C-preincubated Csa microsomes, was inhibited 40% by 0.03 mM propylpyrazole, a potent inhibitor of alcohol dehydrogenase. Due to the presence of peroxidatically-uncoupled catalase in 43 C-preincubated

Csb microsomes, residual MEOS activity in these preparations was unaffected by 0.1 mM cyanide or azide, but was totally obliterated by 0.03 mM propylpyrazole. In contrast, the same concentration of propylpyrazole produced only a 10% inhibition of NADPH-dependent aniline p-hydroxylation and aminopyrine, ethylmorphine, and benzphetamine *N*-demethylation activities of "zero-time" or of 43 C-preincubated microsomes from either strain. Incubation of Csb microsomes for 10 min at 37 C or 30 C resulted in catalase activities that were 72% and 102%, resp., of those obtained with similarly incubated Csa microsomes. Furthermore, incubation of Csb microsomes at 37 C (10 min) with ethanol and 0.5 or 5.0 µg/ml glucose oxidase gave rise to ethanol oxidation rates that were 65% and 30%, resp., of corresponding values obtained with Csa microsomes. Clearly, inactivation of catalase in Csb microsomes during 37 C incubations causes a shift in the H_2O_2/catalase ratio to higher values, leading to a disproportionately larger contribution of the "catalatic" reaction and a greater inhibition of ethanol oxidation at 5.0 as opposed to 0.5 µg/ml glucose oxidase. In contrast, no differences in ethanol oxidation rates were observed between Csa and Csb microsomes incubated at 30 C (10 min), regardless of glucose oxidase concentration. These studies indicate strongly that hepatic MEOS activity can be accounted for by the concerted action of catalase (60-70%) and alcohol dehydrogenase (30-40%).

8081 METABOLISM OF ALCOHOL AT HIGH CONCENTRATIONS: ROLE AND BIOCHEMICAL NATURE OF THE HEPATIC MICROSOMAL ETHANOL OXIDIZING SYSTEM. (Eng.) Teschke, R.; Matsuzaki, S.; Ohnishi, K.; Hasumura, Y.; Lieber, C. S. (Veterans Admin. Hosp., Bronx, NY 10468). *Adv. Exp. Med. Biol.* 85A:257-280; 1977.

Studies concerning the role and biochemical nature of the hepatic microsomal ethanol oxidizing system (MEOS) are reviewed, and results of the authors' experiments are presented. At intermediate and higher alcohol concentrations, ethanol metabolism proceeds via alcohol dehydrogenase (ADH) and MEOS, whereas catalase plays no significant role. Following prolonged ethanol consumption, an enhancement of both MEOS activity and the rates of ethanol metabolism occurs; the latter persists despite inhibition of ADH by pyrazole and catalase by sodium azide, suggesting that MEOS is involved in the adaptive increase. This conclusion was supported by the authors' experiments in which butanol had no inhibitory effect on *in vitro* oxidation of ethanol mediated by catalase in the presence of a H_2O_2-generating system. In liver slices, the pyrazole-insensitive ethanol oxidation was decreased by 43.2% ($p<0.01$) in the presence of butanol, a value similar to the corresponding result obtained in isolated microsomes for the inhibition of MEOS. These results suggest that the MEOS is responsible for the pyrazole-insensitive pathway. Other experiments in which aminotriazole pretreatment failed to inhibit ethanol metabolism provided strong evidence against a significant involvement of catalase in ethanol metabolism under physiological conditions in which no DL-alanine was added. MEOS exhibits characteristics similar to those of other microsomal drug metabolizing enzymes and can be differentiated and isolated from both ADH and catalase activities. Reconstitution of MEOS activity was achieved with partially purified cytochrome P-450 and NADPH-cytochrome c reductase in the presence of synthetic phospholipid. The results of the present studies and others in the literature have led to the acceptance of the previously proposed thesis that MEOS is responsible for a major part of the adaptive increase in ethanol metabolism following chronic ethanol consumption and that the microsomal system is a distinct mechanism capable of oxidizing various alcohols by a pathway not requiring catalase or ADH.

8082 ALCOHOL METABOLISM AT HIGH ALCOHOL CONCENTRATIONS. (Eng.) Thieden, H. I. D; Quistorff, B.; Grunnet, N. (Dept. Biochemistry A, Panum Inst., Univ. Copenhagen, DK-2200 Copenhagen, Denmark). *Adv. Exp. Med. Biol.* 85A:247-255; 1977.

The incorporation rate of tritium from (R,S)ethanol-1-^{3}H, (R)ethanol-1-^{3}H, and (S)ethanol-1-^{3}H in lactate and β-hydroxybutyrate was investigated by gas-liquid chromatography to estimate the extent of participation of alcohol dehydrogenase in ethanol oxidation at high concentrations of ethanol and to determine the intracellular location of acetaldehyde oxidation. In rat hepatocyte cultures containing 3 mM pyruvate in which 65 mM (R,S)ethanol-1-^{3}H was used as substrate, the labeling rate of lactate declined with time. The rate declined with the decrease in pyruvate concentration in the incubation medium and the concomitant fall in lactate production. The tritium incorporation rate into lactate was decreased from 1.5 µmol/min/ml packed cells to 0.23 µmol/min/ml in the presence of 4-methylpyrazole. In these experiments, the alcohol dehydrogenase-mediated ethanol oxidation rate was 3.18 ± 0.19 µmol/min/g wet weight, indicating that less than 20% of the acetaldehyde is oxidized in the cytoplasm. Using (R)ethanol as substrate, a constant fraction of tritium was incorporated into lactate, indicating that this incorporation is proportional to the formation of ^{3}H-NADH in this compartment. Tritium incorporation into lactate from (R)ethanol did not decrease when the ethanol concentration was increased from 6 mM to 65 mM. Providing the same isotope effect is present at high and low ethanol concentrations, the latter result implies that no substrate inhibition of alcohol dehydrogenase activity occurs in liver cells. Alternatively, a shift in the isotope effect could occur at high ethanol concentrations.

8083 ETHANOL METABOLISM, OXYGEN AVAILABILITY AND ALCOHOL INDUCED LIVER DAMAGE. (Eng.) Israel, Y.; Kalant, H.; Khanna, J. M.; Orrego, H.; Phillips, M. J.; Stewart, D. J. (Dept. Pharmacology,

Univ. Toronto, Ontario M5S 1A8, Canada). *Adv. Exp. Med. Biol.* 85A:343-358; 1977.

Studies concerning ethanol metabolism, oxygen availability, and alcohol-induced liver injury are reviewed. The rates of ethanol oxidation in various species are linked to the rates of general metabolism, and more specifically that of O_2 utilization by the liver. After chronic ethanol administration, increased oxidation of ethanol is accompanied by increased total hepatic O_2 utilization, as studied in the whole animals, the perfused liver, and isolated liver slices. The correlation is particularly striking in the spontaneously hypertensive rat. The linkage of these two phenomena makes the livers of rats chronically consuming ethanol abnormally vulnerable to hypoxic damage. Exposure to a 5% oxygen atmosphere for 6 hr produced major elevations of SGOT and serum ornithine-carbamyl transferase activities and marked centrilobular necrosis in alcohol-treated animals but not in controls. The same differential susceptibility was found to acute anemia produced by bleeding. These data indicate that chronic ethanol administration increases the susceptibility of the liver to ischemic injury.

8084 METABOLISM OF 25-HYDROXYLATED BILE ALCOHOLS. (Eng.) Mosbach, E. H.; Salen, G.; Shefer, S. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* (Baltimore: University Park Press): 11-15; 1977.

Studies concerning 25-hydroxylated bile alcohol metabolism are reviewed. Patients with the sterol storage disease cerebrotendinous xanthomatosis (CTX) secrete 25-hydroxylated bile alcohols in bile and feces. Quantitatively, the predominant bile alcohols are 5β-cholestane-3α,7α,12α,25-tetrol, 5β-cholestane-3α,7α,12α,23β,25-pentol, and 5β-cholestane-3α,7α,12α,24α,25-pentol. CTX patients produce cholic acid from cholesterol mainly via the intermediate 5β-cholestane-3α,7α,12α,25-tetrol, while chenodeoxycholic acid is formed probably via 5β-cholestane-3α,7α,25-triol. Nevertheless, subjects with CTX have the capacity to transform a 26-hydroxylated bile alcohol, 5β-cholestane-3α,7α,-26-triol into cholic acid and chenodeoxycholic acid. Experiments *in vitro* indicate that hepatic microsomal enzymes of CTX patients, rat, and man catalyze the hydroxylation of 5β-cholestane-3α,7α,-12α,25-tetrol in the 24α and 24β position, in the presence of O_2 and NADPH. However, only 5β-cholestane-3α,7α,12α,24β,25-pentol is converted to cholic acid (plus acetone) by soluble hepatic enzymes. This transformation requires NAD and probably involves a 24-ketone as an intermediate.

8085 ACETALDEHYDE METABOLISM IN VIVO DURING ETHANOL OXIDATION. (Eng.) Eriksson, C. J. P. (Res. Lab. State Alcohol Monopoly, Box 350, SF-00101 Helsinki 10, Finland). *Adv. Exp. Med. Biol.* 85A:319-341; 1977.

Information concerning acetaldehyde (AcH) metabolism during ethanol oxidation is reviewed. The liver is the primary site for the oxidation of ethanol-derived AcH in the rat. Only a small amount of the total AcH formed in this organ escapes into the rest of the body, but this amount increases with increasing hepatic ethanol concentrations. The bulk of the hepatic AcH output is eliminated extrahepatically, thus drastically changing the AcH level from that initially leaving the liver. Nevertheless, the extrahepatic blood AcH levels can be used as relatively accurate indicators of the corresponding hepatic AcH levels, since they are highly correlated with them. Significant levels of brain AcH occur only at very high arterial blood AcH concentrations.

8086 THE ACUTE EFFECTS OF ETHANOL ON LIVER AND BRAIN TRYPTOPHAN METABOLISM. (Eng.) Badawy, A. A. B.; Evans, M. (Witchurch Hosp., Cardiff, CF4 7XB, Wales). *Adv. Exp. Med. Biol.* 85A: 559-569; 1977.

The effects of acute ethanol administration on liver and brain tryptophan metabolism are reviewed. Ethanol enhances the activity of rat liver tryptophan pyrrolase by increasing the availability of circulating free tryptophan to the liver by catecholamine-mediated lipolysis followed by displacement of protein-bound serum tryptophan. The response of the mouse liver enzyme to ethanol is strain-dependent; ethanol activates the enzyme in CBA/CA but not in C57/BL mice. Ethanol exerts a biphasic effect on the concentrations of rat brain tryptophan, 5-hydroxytryptamine, and 5-hydroxyindole-3-yl-acetic acid. Both aspects of this biphasic effect are associated with an altered availability of circulating free tryptophan. The initial enhancement by ethanol of brain tryptophan metabolism may be due to the above mentioned lipolytic mechanism, whereas the subsequent decrease in brain indoles may be caused by the enhanced tryptophan pyrrolase activity. Brain tryptophan metabolism is decreased by ethanol in CBA/CA mice, whereas no such change is observed in C57/BL mice.

8087 EFFECT OF THE COENZYMES UDP-Glc, CDP-Ch, SAM AND CoA-SH ON THE LIVERS OF RATS TREATED WITH ETHANOL AND A HIGH-FAT DIET. (Fre.) Cascales, M.; Feijoo, B.; Cerdan, S.; Robles-Chillida, E. M.; Santos-Ruiz, A. (Universidad Complutense, Madrid, Spain). *Ann. Pharm. Fr.* 35(11/12): 447-459; 1977.

The protective effect of the lipotropic substances uridine-diphosphoglucose (UDP-Glc), S-adenosylmethionine (SAM), cytidine-diphosphocholine (CDP-Ch), and coenzyme A (CoA-SH) against development of fatty liver in rats fed ethanol and a high-fat diet was investigated. Rats were given either ethanol (20% in the drinking water) and a diet composed of 45% vegetable fat or a normal diet without ethanol. Half the rats on the high-fat diet with ethanol were injected i.p. daily with 30 mg/kg UDP-Glc, 1.5 mg/kg CDP-Ch, 3 mg/kg SAM, and 0.1 mg/kg CoA-SH, where-

as the other half (controls) were injected daily with an identical volume of physiological saline solution. At the end of 3 months, the rats were killed, and their livers were weighed, examined histologically, and their content of oxidoreductive and urea cycle enzymes determined. Differences in the ratio of liver weight/body weight in treated and untreated rats on the ethanol and high-fat diet were not significant. Untreated rats on the ethanol and high-fat diet had fatty livers. Ultrastructural study of the livers of rats on the high-fat with ethanol diet showed a marked decrease (from 46% to 5%) in the number of hepatocytes with fatty vacuoles in rats treated with lipotropic substances compared with the untreated rats; in the treated rats, fatty hepatocytes were found grouped around the blood vessels. Of the liver oxidoreductive enzymes whose levels were significantly ($p<0.05$-0.001) reduced (glucose-6-phosphate dehydrogenase, 6-phosphogluconate dehydrogenase [6PGDH], transaldolase, and isocitrate dehydrogenase NADP) by the high fat and ethanol regime, only the 6PGDH level was significantly ($p<0.05$) restored by the protective treatment. Of the urea cycle enzymes whose levels were significantly ($p<0.05$-0.001) reduced (carbamyl-phosphate synthetase [CPase], ornithine transcarbamylase, and arginase) by the high-fat and ethanol diet, CPase and arginase were significantly ($p<0.01$) restored by treatment. Liver GOT and glutamate dehydrogenase levels were decreased in rats on the high-fat and ethanol diet ($p<0.05$-0.001); however, administration of the lipotropic agents significantly ($p<0.05$) prevented the decrease of GOT. The lipotropic agents also appeared to prevent the accumulation of free ammonia in the livers of rats on the high-fat and ethanol diet.

8088 THE EFFECT OF PURINE BASES AND NUCLEOTIDES ON ETHANOL-INDUCED FATTY LIVER SYNDROME.

(Eng.) Auditore, J. V.; Crisona, F.; Patthey, H.; Akintonwa, A. (Meharry Medical Coll., Nashville, TN 37208). *Arch. Int. Pharmacodyn.* 232(2):302-308; 1978.

The capacity of the purine bases and their nucleoside derivatives to prevent chronic ethanol-induced fatty liver and depressed adenine nucleotide levels was investigated in female Sprague-Dawley rats. The mean liver triglyceride concentration in rats fed a control diet (12.3 ± 1.1 mg/g wet weight liver) was significantly lower ($p<0.001$) than that in rats fed either a control liquid fat diet (23.9 ± 2.1 mg/g), a fat diet plus adenine as 0.1% of diet (20.1 ± 1.8 mg/g), a fat diet plus 0.1% ATP (21.8 ± 2.0 mg/g), a fat diet plus 0.1% guanine (22.0 ± 1.6 mg/g), a fat diet plus 0.1% GTP (24.0 ± 1.9 mg/g), a fat diet plus ethanol as 20% of caloric intake (72.0 ± 5.1 mg/g), a fat diet plus ethanol with or without adenine (43.7 ± 6.2 mg/g), a fat diet plus guanine plus ethanol (70.1 ± 4.8 mg/g), a fat diet plus ATP plus ethanol (68.6 ± 4.3 mg/g), or a fat diet plus GTP plus ethanol (71.1 ± 5.3 mg/g). The hepatic ATP levels were significantly lower in animals fed the fat diet (2.70 ± 0.06 μmol/g wet weight liver) than in those fed the control diet (2.96 ± 0.08 μmol/g). Ethanol alone depressed the hepatic ATP level more than 85% of normal, and the level in the animals fed the fat diet plus ethanol alone (1.48 ± 0.8 μmol/g) was significantly reduced compared with the levels in animals fed control diet, fat diet, fat diet plus adenine (2.80 ± 0.08 μmol/g), fat diet plus ATP (2.76 ± 0.05 μmol/g), fat diet plus guanine (2.67 ± 0.09 μmol/g), fat diet plus GTP (2.60 ± 0.10 μmol/g), or fat diet plus ethanol plus adenine (2.32 ± 0.12 μmol/g). The incorporation of adenine into the fat diet partially restored hepatic ATP levels, and a slight increase was observed with ATP in the diet, but these differences were not statistically significant. Guanine and GTP failed to restore normal ATP levels.

8089 STUDIES ON FATTY LIVER WITH ISOLATED HEPATOCYTES. I. THE ACTION OF COLCHICINE, PHALLOIDIN, CYTOCHALASIN B, AND CYCLOHEXIMIDE ON PROTEIN AND TRIGLYCERIDE SYNTHESIS AND SECRETION.

(Eng.) Gravela, E.; Poli, G.; Albano, E.; Dianzani, M. U. (Istituto di Patologia Generale, Corso Raffaello 30, 10125 Torino, Italy). *Exp. Mol. Pathol.* 27(3):339-352; 1977.

The effects of colchicine, phalloidin, cytochalasin B, and cycloheximide on protein and triglyceride synthesis and secretion were studied in isolated rat hepatocytes to investigate the role of derangement of contractile protein systems in the pathogenesis of fatty liver. Control cells and cells treated with 20 μM colchicine or cycloheximide were completely dissociated from each other and demonstrated intact cell membranes. Five minutes after the addition of 20 μM cytochalasin B, plasma membranes showed numerous bubbles and many hepatocytes formed clusters of 4-10 cells. Similar changes were seen with 20 μM phalloidin, but these occurred less rapidly (after about 60 min). Colchicine (10 μM) produced 35% inhibition of protein secretion after 60 min of incubation; after 90 and 120 min, it reached about 60-65% inhibition. Cycloheximide (0.7 μM) produced a 20% decrease in protein secretion aft r 30 min, and a 40% decrease was noted after 60 min; inhibitions after 90 and 120 min were 55% and 60%, resp. Phalloidin (20 μM) had no effect on protein secretion after 30 min but provoked 50-60% inhibition at 60-120 min. While cytochalasin B (20 μM) did not significantly inhibit protein secretion after 30 min, about 50% inhibition occurred after 60 min; inhibition decreased slightly thereafter. Colchicine (10 μM) inhibited lipoprotein triglyceride secretion by about 60% at 30 min. Cycloheximide (0.7 μM) had no effect at 30 min but inhibited triglyceride secretion by 40% after 60-120 min. Phalloidin (20 μM) inhibited triglyceride secretion by 35% after 30 min, by 50% after 60 and 90 min, and by 40% by 120 min. Cytochalasin B (20 μM) did not modify triglyceride secretion at any time. There was a good quantitative correspondence between the decrease in secreted triglyceride radioactivity and the increase in intracellular triglyceride radioactivity, indicating that colchicine, cycloheximide, and phalloidin produced *in vitro* liver steatosis. The substances studied did not significantly modify triglyceride synthesis. The results show that

protein and lipoprotein secretion from isolated liver cells may be blocked in at least two ways: (a) by inhibition of protein synthesis and (b) by impairment of the microtubular system.

8090 D-GALACTOSAMINE INDUCED HEPATIC CIRRHOSIS: ITS ULTRASTRUCTURAL AND BIOCHEMICAL STUDIES IN RAT. (Eng.) Funatsu, K.; Ishii, H.; Shigeta, Y.; Morita, A.; Tsuchiya, M. (Sch. Medicine, Keio Univ., Shinjuku-ku, Tokyo 160, Japan). *Acta Hepatogastroenterol.* 25(2):97-104; 1978.

Ultrastructural and biochemical studies of the liver were carried out in female Wistar rats treated with D-galactosamine (GALN, 250 mg/kg, i.p.) for 7 months. Histological examination of liver specimens taken at the end of treatment showed cirrhotic changes in most animals. Ultrastructural study showed mitochondrial proliferation and crenation of the mitochondrial membranes, focal hypertrophy of the smooth endoplasmic reticulum, saccular dilatation of the rough endoplasmic reticulum, the absence of liposomes in dilated tubules of the smooth endoplasmic reticulum, and smooth scattered fat droplets in the cytoplasm. No large fat droplets were observed in the cytoplasm. The nucleolous was relatively well-preserved, contrasting with the fact that marked fragmentation or small spherical deformities of the nucleoli are characteristic of GALN-induced liver injury. Proliferation of collagen fibers was present around the hepatocytes, and ruthenium red-positive material was found in the spaces of Disse and in part of the sinusoids. Liver weight increased significantly ($p<0.01$) after GALN treatment. However, the total lipid, triglyceride, and phospholipid concentrations were not affected by the treatment. There was a significant decrease in microsomal protein concentration after treatment ($p<0.005$) and an increase in the cytochrome P450 content ($p<0.05$). The plasma albumin concentration was significantly decreased after treatment ($p<0.01$), and the value of the zinc turbidity test was increased ($p<0.05$). There was no change in the SGPT level or in the total cholesterol concentration. Since there was no change in the contents of total hepatic lipids, triglycerides, proteins, or phospholipids, the increased liver weight after GALN treatment may be due to an increase in the amount of liver collagen fibers. It is unlikely that chronic GALN administration produces smooth endoplasmic reticulum hypertrophy in the rat liver, since there was no significant change in the microsomal phospholipid concentration and since the microsomal protein content was decreased.

8091 MECHANISM OF PALMITYL COENZYME A INHIBITION OF LIVER GLYCOGEN SYNTHASE. (Eng.) Wititsuwannakul, D.; Kim, K. H. (Dept. Biochemistry, Purdue Univ., West Lafayette, IN 47907). *J. Biol. Chem.* 252(21):7812-7817; 1977.

8092 RAT LIVER GLUCOSE-6-P DEHYDROGENASE: DIETARY REGULATION OF THE RATE OF SYNTHESIS. (Eng.) Winberry, L.; Holten, D. (C. H. Best Inst., 112 College St., Univ. Toronto, Toronto, Ontario M56 1L6, Canada). *J. Biol. Chem.* 252(21):7796-7801; 1977.

8093 PURINE METABOLISM IN ISOLATED RAT HEPATOCYTES. (Eng.) Smith, C. M.; Rovamo, L. M.; Kekomaki, M. P.; Raivio, K. O. (Children's Hosp., SF-00290 Helsinki 29, Finland). *Can. J. Biochem.* 55(11):1134-1139; 1977.

8094 INTERRELATION OF AEROBIC GLYCOLYSIS AND LIPOGENESIS IN ISOLATED PERFUSED LIVER OF WELL-FED RATS. (Eng.) Walli, R. A. (Institut fur Biochemie I der Universitat Heidelberg, Im Neuenheimer Feld 328, 69 Heidelberg, W. Germany). *Biochim. Biophys. Acta* 539(1):62-80; 1978.

8095 BILE SALT METABOLISM. I. THE PHYSIOLOGY OF BILE SALTS. (Eng.) Cowen, A. E.; Campbell, C. B. (Royal Brisbane Hosp., Brisbane, Queensland, Australia). *Aust. N.Z. J. Med.* 7(6):579-586; 1977.

8096 TEMPERATURE DEPENDENCE OF PHALLOIDIN RESPONSE IN ISOLATED RAT HEPATOCYTES. (Eng.) Frimmer, M.; Rufeger, U. (Fachbereich Veterinarmedizin der Justus Liebig-Universitat Giessen, D-6300 Giessen, W. Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 300(2):193-194; 1977.

8097 TOTAL HEPATIC ISCHAEMIA IN THE RHESUS MONKEY. (Eng.) McMaster, P.; Medd, R. (Addenbrooke's Hosp., Cambridge, England). *Br. J. Surg.* 64(11):819-821; 1977.

8098 EFFECTS OF ACUTE, PASSIVE HEPATIC CONGESTION ON BLOOD FLOW AND OXYGEN UPTAKE IN THE INTACT LIVER OF THE CAT. (Eng.) Lautt, W. W. (Coll. Medicine, Univ. Saskatchewan, Saskatoon, Saskatchewan S7N OWO, Canada). *Circ. Res.* 41(6):787-790; 1977.

8099 MACROPHAGES AND AGE-DEPENDENT RESISTANCE TO HEPATITIS INDUCED BY HERPES SIMPLEX VIRUS TYPE 2 IN MICE. (Eng.) Mogensen, S. C. (Inst. Medical Microbiology, Univ. Aarhus, DK-8000 Aarhus C, Denmark). *Infect. Immun.* 19(1):46-50; 1978.

8100 Entamoeba histolytica AND Entamoeba invadens: CHROMIUM RELEASE FROM LABELED HUMAN LIVER CELLS IN CULTURE. (Eng.) McCaul, T. F.; Poston, R. N.; Bird, R. G. (Natl. Inst. Health's Rocky Mountain Lab., Hamilton, MN 59840). *Exp. Parasitol.* 43(2):342-352; 1977.

8101 CANINE AND HUMAN LIVER PRESERVATION FOR 6 TO 18 HR BY COLD INFUSION. (Eng.)

Benichou, J.; Halgrimson, C. G.; Weil, R., III; Koep, L. J.; Starzl, T. E. (Denver Veterans Admin. Hosp., Denver, CO). *Transplantation* 24(6):407-411; 1977.

8102 REPEATED ARTERIAL EMBOLIZATION OF RAT LIVERS BY DEGRADABLE MICROSPHERES. (Eng.) Lindell, B.; Aronsen, K. F.; Rothman, U. (Malmo General Hosp., Malmo, Sweden). *Eur. Surg. Res.* 9(5):347-356; 1977.

8103 LIVER PATHOLOGY IN YOUNG RATS FED A NATURAL LYSINE-IMBALANCED DIET. (Eng.) de Parada, N. M.; Garcia de Davila, M. T.; Rio, M. E.; Koch, O. R. (Facultad de Farmacia y Bioquimica, Universidad de Buenos Aires, Junin 956, 1113 Buenos Aires, Argentina). *Nutr. Rep. Int.* 17(2):197-207; 1978.

8104 AN IN VIVO INVESTIGATION OF THE NEGATIVE CHRONOTROPIC EFFECT OF CHOLIC ACID IN THE RAT. (Eng.) Joubert, P. (Dept. Pharmacology, Univ. Orange Free State, P.O. Box 339, Bloemfontein, 9300, South Africa). *Clin. Exp. Pharmacol. Physiol.* 5(1):1-8; 1978.

8105 MICRODENSITOMETRIC AND AUTORADIOGRAPHIC COMPARISON OF THE DNA CONTENTS OF FOETAL AND ADULT RAT LIVER NUCLEI. (Eng.) Bedi, K. S.; Goldstein, D. J. (Medical Sch., Stopford Building, Univ. Manchester, Manchester, England). *Histochemistry* 55(1):63-74; 1978.

8106 COBALT-ACTIVATED ACYLASE ACTIVITY IN EXPERIMENTAL TOXIC LIVER DAMAGE. (Eng.) Sledzinski, Z.; Kokot, F. (Silesian Medical Acad., 40-027 Katowice, Poland). *Arch. Immunol. Ther. Exp.* 25(4):601-610; 1977.

8107 EFFECT OF IRRADIATION ON THE ERYTHROCYTE MEMBRANES OF HOMOZYGOUS GUNN RAT SUCKLINGS. (Eng.) Sato, H.; Katoh-Semba, R.; Kashiwamata, S. (Inst. Developmental Res., Aichi Prefectural Colony, Kamiya-cho, Kasugai, Aichi 480-03, Japan). *Biol. Neonate* 32(3/4):158-165; 1977.

8108 PATHOGENESIS AND ROLE OF MICROTUBULAR ALTERATIONS IN ALCOHOL INDUCED LIVER INJURY [Abstract]. (Eng.) Matsuda, Y.; Baraona, E.; Salaspuro, M.; Lieber, C. S. (Bronx Veterans Admin. Hosp., Bronx, NY). *Fed. Proc.* 37(3):402; 1978.

8109 INHIBITION OF DIETHYLNITROSAMINE (DEN) INDUCED ACUTE LIVER CELL NECROSIS BY DIETHYLDITHIOCARBAMATE (DEDTC): A POSSIBLE SITE OF ACTION AFTER THE ACTIVATION STEP [Abstract]. (Eng.) Ying, T. S.; Sarma, D. S. R.; Farber, E. (Dept. Pathology, Univ. Toronto, Toronto, Ontario, Canada). *Fed. Proc.* 37(3):402; 1978.

8110 CHOLERETIC POTENCY OF GASTRIN I (GI) AND GASTRIN II (GII) [Abstract]. (Eng.) Deshpande, Y. G.; Kaminski, D. L. (Dept. Surgery, Saint Louis Univ., St. Louis, MO 63104). *Fed. Proc.* 37(3):697; 1978.

8111 CHARACTERISTICS OF THE HEPATIC TRANSPORT OF THE FOLATE ANALOG, AMETHOPTERIN [Abstract]. (Eng.) Strum, W. B.; Liem, H. H.; Muller-Eberhard, U. (Scripps Clinic, La Jolla, CA). *Clin. Res.* 26(2):113A; 1978.

8112 STUDIES OF BILE SALT TRANSPORT: EFFECT OF CYCLOHEXIMIDE ON HEPATIC BILE SALT TRANSPORT AND BINDING [Abstract]. (Eng.) Gonzalez, M. C.; Sutherland, E.; Simon, F. R. (Univ. Colorado Medical Center, Denver, CO). *Clin Res.* 26(2): 110A; 1978.

8113 SEX DIFFERENCES IN HEPATIC SULFATION OF TAUROLITHOCHOLATE [Abstract]. (Eng.) Hammerman, K.; Chen, L.; Fernandez, A.; Earnest, D. (Dept. Medicine, Univ. California, San Francisco, CA). *Clin. Res.* 26(2):110A; 1978.

See also, 7982, 7985, 7997, 7998, 8038, 8047, 8061, 8139, 8244, 8439, 8440, 8441, 8442, 8444, 8470, 8516, 8526, 8534, 8558.

SECRETION AND METABOLISM

Intestine

8114 USE OF THE CONSTANT PERFUSION TECHNIQUE IN THE NONSTEADY STATE. (Eng.) Levitt, M. D.; Bond, J. (Section Gastroenterology, Box 346 Mayo, Univ. Minnesota, Minneapolis, MN 55455). *Gastroenterology* 73(6):1450-1453; 1977.

The source and magnitude of errors that may occur in measurements during nonsteady-state perfusion (e.g., gallbladder or gastric emptying) are presented. Errors arise (1) when the aliquots taken from two compartments of unequal volume are pooled for analysis but were not proportional in size to their respective compartment volumes (the total volume of the two compartments is underestimated; the

error is nearly 50% when one compartment exceeds the other by a factor of 5 in volume), (2) when aliquots aspirated intermittently at a constant rate from a compartment of inherently fluctuating volume are pooled for analysis (if the volume changes instantaneously by a factor of 5, there is the same error and magnitude as in [1] above), (3) when the error committed in (2) is exacerbated by measuring the infusate volume as well (the underestimation in [1] is increased to >80%), (4) when one fraction of the aspirate has a higher marker concentration than does the remainder of the aspirate due to variations in the flow rate of perfusate and the fractions are pooled for analysis (if the marker concentrations is overestimated, then the true flow rate will be underestimated and vice versa, the magnitude of the error depending on the fluctuation of the marker concentration in the system and the relation of the aspiration rate to the true flow rate), (5) if a higher concentration of solute exists during periods of high flow and samples from different flow periods are pooled for analysis (the concentration of the solute and, hence, its absorption or secretion flow rate, is underestimated to an even greater extent than is volume), and (6) if a lower concentration of solute is present during periods of high flow and the samples are pooled for analysis (the marker concentration is overestimated, but the underestimation of volume flow reduces the error in the calculated total solute flow rate). Under nonsteady-state conditions, analysis of repeated small aliquots of aspirate would provide some information on the magnitude of the error, although precise calculation is impossible unless the fraction of perfusate aspirated is known. When constant-rate aspiration introduces error into the measurement of the flow rates of solutes not in the perfusate, the fastest feasible infusion rate should provide the most accurate result; if infusion must be slow, short-interval aspiration will reduce error. Also, the maximum possible quantity of perfusate should be collected as opposed to aspiration at a fixed rate. Two situations where a correct estimate of solute flow may be obtained despite nonsteady conditions are (1) if the test solute enters and is absorbed into the test segment at a constant rate independent of volume flow and (2) if the marker concentration is constant but solute concentration fluctuates.

8115 CHRONIC ALCOHOL CONSUMPTION DOES NOT MODIFY CHOLECYSTOKININ BLOOD LEVELS ESTIMATED BY BIOASSAY IN THE DOG. (Eng.) Planche, N. E.; Chau-Huu, T.; Lai, P. P.; Sarles, H. (Unite de Recherches de Pathologie Digestive, Inserm U-31, 46, chemin de la Gaye, F-13009, Marseilles, France). *Digestion* 16(1/2):194-198; 1977.

To examine the hypothesis that increased pancreatic protein secretion in chronic alcoholism is due to increased cholecystokinin (CCK) release, blood levels of CCK were measured by an *in vitro* rabbit gallbladder bioassay in two series of dogs: five that received 2 g/kg 45% ethanol/day for 12-18 months via a gastric cannula and five controls. There was no difference in CCK activity between the two groups, either in the fasting state, after intragastric administration of ethanol, or after intraduodenal administration of 20 ml oleic acid following the infusion of secretion (1 clinical U/kg/hr, i.v., for 1 hr). CCK release is not modified during the course of chronic alcohol intoxication in the dog, and, therefore, differences in CCK release do not play a part in increased pancreatic protein secretion in chronic alcoholic dogs.

8116 TRANSFER AND METABOLISM OF CITRATE, SUCCINATE, α-KETOGLUTARATE AND PYRUVATE BY HAMSTER SMALL INTESTINE. (Eng.) Browne, J. L.; Sanford, P. A.; Smyth, D. H. (Middlewood Hosp., Sheffield, England). *Proc. R. Soc. Lond.* [*Biol.*] 200(1139):117-135; 1978.

The transfer of citrate, succinate, α-ketoglutarate, and pyruvate was studied in everted sacs of hamster small intestine to determine specific transfer mechanisms for each of these. All four intermediates were rapidly removed from the mucosal fluid (14.0, 11.7, 8.9, and 9.2 μmol/hr for succinate, α-ketoglutarate, citrate, and pyruvate, resp.) and appeared in the serosal fluid in the presence of metabolites. Only in the case of citrate, where consistently high recoveries of the unchanged intermediate were obtained, was there chromatographic evidence for transfer against a concentration gradient. The inhibitory effects of several carboxylates, some of which are not intermediates of the citric acid cycle, were tested. All the dicarboxylates tested (succinate, α-ketoglutarate, fumarate, oxaloacetate, glutarate, and malate, all at 7.5 mM), with the exception of the latter two, were potent inhibitors (by >70%) of citrate transfer. Tricarballylate and pyruvate (both 7.5 mM) also produced significant inhibition (69% and 26%, resp.), but the inhibition produced by 7.5 mM *cis*-aconitate was insignificant. The dicarboxylates produced inhibitions of succinate transfer that were significant at the 5% level, although less than the inhibitions produced on citrate transfer (5-82%). The 5% inhibition seen with glutamate was the only insignificant inhibition. Citrate, tricarballylate, and pyruvate reduced transfer by 54%, 59%, and 36%, resp., all significant at the 5% level; the effect of *cis*-aconitate was again insignificant. A similar pattern of inhibition of α-ketoglutarate transfer was seen, in that the effects of the dicarboxylates were greatest while the effect of pyruvate was least. These results suggest that carboxylates compete at the luminal membrane for the mechanisms involved in citrate, succinate, and α-ketoglutarate transfer, and that the mechanism for the transfer of these intermediates is different from the pathway involved in pyruvate transfer. The transfer processes for citrate and succinate were saturable, and more than one pathway may be involved in the transfer of succinate. Pyruvate showed little interaction with the other three intermediates. A saturable component of transfer was demonstrated, although diffusion contributed a considerable proportion of the total transfer. The

importance of a downhill sodium gradient for transfer of citrate, succinate, α-ketoglutarate, and pyruvate across the luminal membrane was established. A number of carboxylates stimulated the potential differences across the intestine, although this was different from the potential difference produced by sugars and amino acids.

8117 INFLUENCE OF ANTIBIOTICS ON MICROBIAL INTESTINAL TRANSFORMATION OF CHOLESTEROL TO COPROSTANOL IN MAN. (Eng.) Midvedt, T.; Frederichsen, P. (Kaptein W. Wilhelmsen og Frues Bakteriologiske Institutt, Univ. Oslo, Oslo, Norway). *Scand. J. Gastroenterol.* 12(6):669-672; 1977.

The fecal excretion of coprostanol was studied in 19 healthy volunteers receiving antibiotics to investigate the role of intestinal bacteria in the conversion of cholesterol to coprostanol. The following antibiotics were used: doxycycline (4 subjects), oxitetracycline (3), ampicillin (3), phenoximethylpenicillin (3), clindamycin (3), neomycin sulfate (1), and bacitracin (2). Before antibiotic administration, coprostanol excretion far exceeded that of cholesterol in all but one subject. Antibiotic administration reduced the coprostanol/cholesterol ratio to less than 1 in all cases except subjects treated with doxycycline throughout a 3-week observation period. In subjects that received phenoximethylpenicillin, the ratio went below 1 the day after administration of the drug was discontinued in one of the three men investigated, increasing to 3.0 and 6.9, resp., 1 and 2 weeks later. In subjects receiving clindamycin, ampicillin, or tetracycline, no coprostanol was detected on the first day, and in two of them, production of coprostanol was absent even after 12 and 15 weeks, resp. These results show that four of six antibiotics tested have a marked influence on microbial intestinal cholesterol transformation to coprostanol. Further studies are required to determine whether an antibiotic-induced absence of intestinal coprostanol production may have an influence on other aspects of cholesterol metabolism.

8118 HORMONAL STIMULATION OF INTESTINAL BRUSH BORDER ENZYMES RELEASE. (Eng.) Eloy, R.; Vaultier, J. P.; Raul, F.; Mirhom, R.; Clendinnen, G.; Grenier, J. F. (Unit 61 INSERM, Surgical Res. and Biophysiopathology of the Intestine, Avenue Moliere, F-67200 Strasbourg, France). *Res. Exp. Med. (Berl.)* 172(2):109-121; 1978.

The importance of intestinal brush border enzyme release in response to graded doses of glucagon, pentagastrin, and cerulein was studied in rats after common bile duct and pancreatic duct ligation. The intraluminal release of sucrase, alkaline phosphatase, aminopeptidases, and enterokinase was increased after the *in vivo* administration of any one of the hormones at doses ranging between 10 pg and 2 μg, with some changes reaching significance ($p<0.005$-0.05). *Ex vivo* perfusion with glucagon (2 ng) significantly increased the activities of sucrase ($p<0.005$), alkaline phosphatase ($p<0.01$), and aminopeptidase ($p<0.05$) above control values. *Ex vivo* perfusion with 20 or 200 pg pentagastrin significantly elevated the activities of sucrase, alkaline phosphatase, and aminopeptidase above control levels ($p<0.005$-0.05 at various times). The *ex vivo* administration of cerulein (100 pg) significantly stimulated sucrase, alkaline phosphatase, and aminopeptidase activities ($p<0.005$-0.05). The results suggest that there is a direct hormonal stimulation of the intestinal mucosa. This effect might be mediated, at least in part, by cyclic AMP, since 2 mM dibutyryl cyclic AMP exerted a marked stimulatory effect on the intraluminal release of proteins, sucrase, and enterokinase; however, the enzyme pattern following this stimulation was different from that produced by the three hormones.

8119 FOLATE CONJUGASE: TWO SEPARATE ACTIVITIES IN HUMAN JEJUNUM. (Eng.) Reisenauer, A. M.; Krumdieck, C. L.; Halsted, C. H. (Dept. Internal Medicine, Univ. California, Davis, CA 95616). *Science* 198(4313):196-197; 1977.

Folate conjugase activity from jejunal segments obtained at surgery in obese patients undergoing jejuno-ileal bypass was assayed using cell fractionation and diethylaminoethyl (DEAE)-cellulose and gel chromatography. On the basis of pH optima, inhibition by *p*-hydroxymercuribenzoate (PHMB), and molecular weight, two separate jejunal mucosal folate conjugase activities were found. One enzyme activity, membrane-bound and concentrated in the brush border fraction, was found to have an approximate molecular weight of 91,000 daltons, a neutral pH optimum, and no apparent requirement for sulfhydryl groups in the active site, since it was not inhibited by PHMB. The second enzyme activity was soluble and intracellular, had an approximate molecular weight of 45,000 daltons, an acid pH optimum, and an apparent requirement for sulfhydryl groups, since it was completely inhibited by PHMB. Although certain characteristics of the brush border and supernatant folate conjugase activities may have been altered by detergent treatment, nevertheless, prior to Triton solubilization, the brush border and supernatant fractions obtained by centrifugation exhibited distinct differences with regard to pH optima and PHMB inhibition. Although precise localization was not attempted, it is probable that the intracellular activity represents previously described lysosomal folate conjugase. Folate conjugase in the brush border may accomplish the initial digestion of dietary pteroylpolyglutamates.

8120 TOPOGRAPHICAL STUDIES ON INTESTINAL MICROVILLOUS LEUCINE β-NAPHTHYLAMIDASE ON THE OUTER MEMBRANE SURFACE. (Eng.) Takesue, Y.; Nishi, Y. (Res. Inst. Environmental Medicine, Nagoya Univ., Nagoya, Aichi 464, Japan). *J. Membr. Biol.* 39(4): 285-296; 1978.

The distance that the leucine β-naphthylamidase (LNAase) protrudes from the outer surface of the microvillous membrane was investigated in the rabbit small intestine, using an immunological technique combined with electron microscopy. Microvillous vesicles were incubated with various

amounts of preparations of IgG against LNAase (anti-L IgG) at 37 C for 60 min and then at 4 C for 20 hr or for 3 days. Vesicle agglutination was then determined by LNAase or sucrase assay methods. At the same time, inhibition of LNAase activity by anti-L IgG was measured. Microvillous vesicles with an LNAase:sucrase (L:S) ratio of 1.62 were quantitatively agglutinated by anti-L IgG during 20-hr incubation at 4 C, but vesicle agglutination did not occur in microvillous vesicles with an L:S ratio of 0.32, even after 3 days. LNAase inhibition by anti-L IgG was inhibited to a similar degree in both microvillous preparations. The extent of agglutination and inhibition attained by 15 µl of anti-L IgG per unit of LNAase after 20-hr incubation at 4 C were plotted against the L:S ratios of the vesicle preparations. About 90% or more agglutination was observed with vesicles having an L:S ratio higher than 1.4. Agglutination decreased hyperbolically when the L:S ratio was below this value. LNAase inhibition by anti-L IgG was independent of the L:S ratio and was about 65% over the range of L:S ratios examined. In contrast to anti-L IgG, IgG against sucrase-isomaltase complex (anti-SI IgG) quantitatively agglutinated vesicles with a high L:S ratio of 2.26, i.e., rich in LNAase and deficient in sucrase-isomaltase complex, as well as those with a low L:S ratio (0.32). Microvillous vesicles with an L:S ratio of 1.0 were seen ultrastructurally to be closed by the trilaminar membrane with a smooth surface. When these vesicles were incubated with anti-L IgG, continuous or bunch-like structures, appeared on the outer membrane; these protruded 16 nm from the surface. Negative staining showed a 15-nm wide particulate layer on the edge of untreated vesicles; this layer was not broadened by treatment with anti-L IgG. A comparison of these results with previous results in which vesicles were treated with anti-SI IgG has led the authors to conclude that LNAase, or at least its antigenic sites, protrudes about 10 nm from the outer surface of the microvillous membrane.

8121 DISACCHARIDASE AND LYSOSOMAL ENZYME ACTIVITIES IN AMNIOTIC FLUID, INTESTINAL MUCOSA AND MECONIUM: CORRELATION BETWEEN MORPHOLOGY AND DISACCHARIDASE ACTIVITIES IN HUMAN FETAL SMALL INTESTINE. (Eng.) Antonowicz, I.; Milunsky, A.; Lebenthal, E.; Schwachman, H. (Harvard Medical Sch., Boston, MA). *Biol. Neonate* 32(5/6):280-289; 1977.

The relation of sucrase, maltase, and lysosomal enzyme activities to gestational age was investigated in amniotic fluid specimens obtained from 126 women between 14 and 28 weeks of pregnancy. In addition, the morphological development of intestinal mucosa was correlated with sucrase and maltase activities during gestation and the distribution of the brush border membrane-bound and lysosomal enzyme activities in amniotic fluid, intestinal mucosa, and meconium was analyzed in human fetal intestinal mucosa and meconium. Sucrase and maltase activity, which had a relatively constant level between the 14th and 18th weeks, showed a declining pattern thereafter, reaching a very low level at the 26th week of gestation. No lactase activity could be detected between 14 and 26 weeks of gestation. The lysosomal enzymes, α-glucosidase and β-D-fucosidase, showed patterns similar to the activities of sucrase and maltase. No marked changes in *N*-acetyl-β-glucosaminidase activity were noted between weeks 14 and 23. Between the 11th and 20th weeks, the primitive villi seen in sections of small intestine achieved more uniform shape and the crypts became deeper. The activities of intestinal sucrase and maltase at the 10th week were relatively low. Starting with 12th week, these activities increased, and remained approximately the same until the 26th week. At term, the mucosa was completely mature, and the villus to crypt ratio was about 6 to 1; the activities of maltase and sucrase at this time reached the values found in babies <1 yr of age. The ratio of the activities of sucrase and maltase in intestinal mucosa to that in amniotic fluid was relatively constant between the 14th and 18th weeks, being between 5 and 8 times higher in intestinal mucosa than in amniotic fluid. After the 18th week, disaccharidase activities decreased to a very low value in amniotic fluid, while in the intestinal mucosa they remained approximately at the same level. The highest dissacharidase levels were found in fetal meconium from two fetuses 17 and 18 weeks old. The ratio of α-glucosidase in amniotic fluid to intestinal mucosa differed from that of sucrase and maltase being approximately 1:1. The distribution of β-D-fucosidase activity differed considerably from that of α-glucosidase at 17 and 18 weeks. The differences in distribution of the disaccharidases and lysosomal α-glucosidase in amniotic fluid and intestinal mucosa suggest that these enzymes originate in diverse fetal tissues.

8122 EFFECT OF LACTOSE FEEDING ON CELL RENEWAL, DISACCHARIDASE ACTIVITY AND CALCIUM-BINDING PROTEIN CONTENT IN THE INTESTINAL MUCOSA OF RATS. (Eng.) Pansu, D.; Bellaton, C.; Bosshard, A. (Hopital Edouard Herriot, 69374 Lyon Cedex 2, France). *Ann. Biol. Anim. Biochim. Biophys.* 18 (1):127-132; 1978.

To determine how lactose increases the absorption and retention of calcium in the skeleton, intestinal mucosal structure and the levels of disaccharidase activity, calcium binding protein (CaBP), and calcium in the intestinal mucosa were studied in male Wistar rats fed lactose (30% of diet) from weaning. Rats were killed at the age of 4 months for enzymatic and autoradiographic studies, at 2, 3, 4, and 5 months for the CaBP assay, and at 4 and 12 months for skeletal calcium measurement. Prolonged lactose feeding induced significant cecal hypertrophy without mucosal hyperplasia. The cecal weight increased, but mucosal weight and DNA content were similar in the lactose-fed and control rats. Labeling and mitotic indices were also unaltered. The specific activity of invertase was significantly higher in the duodenum (36.1 ± 2.33 mU/mg protein) and cecum (0.46 ± 0.2 mU/mg) of lactose-fed rats than in control rats

(23.9 and 0.23 ± 0.1 mU/mg, resp., $p<0.05$). The CaBP content decreased with age in the duodenum of controls; it was present in the jejunum and ileum (20.40 and 5.0 nmol calcium bound/mg protein, resp.) at 2 months of age but disappeared at 3 months. When a diet containing lactose plus vitamin D (50 IU/100 g) was given, CaBP content decreased at all levels of the intestine and disappeared from the duodenum when the rats were 5 months old. The total body calcium content in 4-month-old lactose-fed rats was similar to that in controls. However, the calcium content was markedly higher (1.23 mg/100 g) in 12-month-old, lactose-fed rats than in controls (1.0 g/100 g). Lactose administration caused no changes in the small intestine that could explain the increased absorption and retention of calcium. The decrease in CaBP content indicates that lactose increases absorption by a mechanism that is not related to active vitamin-dependent transfer.

8123 STUDIES ON THE MEDIATION OF THE ATROPINE-RESISTANT RESPONSE TO NEUROSTIMULATION OF THE GUINEA-PIG DISTAL COLON [Abstract]. (Eng.) Eaglesom, C. C.; Zeitlin, L. J. (Dept. Physiology, Univ. Strathclyde, Glasgow G1 1XW, Scotland). *J. Physiol. (Lond.)* 275:68P-69P; 1978.

8124 INCORPORATION OF 14 ^{14}C-ERUCIC ACID INTO THE INTESTINAL MUCOSA OF MAN AND RAT [Abstract]. (Eng.) Sueur, R.; Cerf, M.; Di Costanzo, G.; Debray, C. (Hopital Bichat, 170, Bld. Ney, 75877 Paris, Cedex 18, France). *Digestion* 16(1/2): 219; 1977.

8125 MICROVISCOSITY OF INTESTINAL CELL SURFACES DURING POSTNATAL DEVELOPMENT [Abstract]. (Eng.) Whitney, J. O.; Hirschberger, M.; Thaler, M. M. (Dept. Pediatrics, Univ. California, San Francisco, CA). *Clin. Res.* 26(2):174A; 1978.

8126 THE INFLUENCE OF RAT AGE ON CRYPT CELL KINETICS AFTER PARTIAL RESECTION OF THE SMALL INTESTINE. (Eng.) Kirchner, F. R.; Bybee, J. W.; Osborne, J. W. (Radiation Res. Lab., Univ. Iowa, Iowa City, IA 52242). *Proc. Soc. Exp. Biol. Med.* 157(4):572-575; 1978.

8127 FUNCTIONAL AND STRUCTURAL CHARACTERISTICS OF THE RAT INTESTINAL MUCOSA FOLLOWING ILEO-JEJUNAL TRANSPOSITION. (Eng.) Menge, H.; Robinson, J. W. L. (Medizinische Universitatsklinik, D-3550 Marburg/Lahn, W. Germany). *Acta Hepatogastroenterol. (Stuttg.)* 25(2):150-154; 1978.

8128 INTESTINAL MUCINS IN HEALTH AND DISEASE. (Eng.) Forstner, J. F. (Hosp. Sick Children, 555 University Ave., Toronto, Ontario M5G 1X8, Canada). *Digestion* 17(3):234-263; 1978.

8129 HORMONE-SENSITIVE ADENYLATE CYCLASE IN HUMAN COLONIC MUCOSA. (Eng.) Simon, B.; Czygan, P.; Spaan, G.; Dittrich, J.; Kather, H. (Medizinische Klinik, D-6900 Heidelberg, W. Germany). *Digestion* 17(3):229-233; 1978.

See also, 7975, 7977, 7984, 7986, 7997, 8004, 8019, 8033, 8040, 8132, 8133, 8134, 8135, 8136, 8168, 8169, 8303, 8353.

GENERAL

8130 MODELING THE GASTROINTESTINAL SYSTEM. (Eng.) Finkelstein, S. M.; Dreiling, D. A.; Blesser, W. B. (Mount Sinai Sch. Medicine, City Univ. New York, New York, NY). *Bull. N.Y. Acad. Med.* 54(3):251-260; 1978.

A basic block diagram model was developed to describe the overall operation of the gastrointestinal system and to study the interaction between the various organs of the system during digestion. The primary blocks of the system represent the stomach, duodenum, pancreas, and gallbladder. Excitation signals for the system are the neural (vagus) responses to food intake and the volume effects of the constituents of food, particularly fat, carbohydrate, and protein. The initial study proposes that both the pH and the volume of the ingested foodstuff stimulate the primary system blocks. The model describes the acid and gastrin responses of the stomach wall, the duodenal hormonal response to the secretin concentration, and the gallbladder response to cholecystokinin in a total closed-loop configuration. Once the model has been validated and quantitative values have been assigned to the various blocks, the model can be simulated on a digital computer and then can be used to study the interactive effect of each subsystem on total system function and the sensitivity of the system's response to variations in parameters.

8131 INFLUENCE OF BODY POSITION, DRY AND WATER SWALLOWS, SMOKING, AND ALCOHOL ON ESOPHAGEAL ACID CLEARING. (Eng.) Kjellen, G.; Tibbling, L. (Univ. Hosp., Linkoping, Sweden). *Scand. J. Gastroenterol.* 13(3):283-288; 1978.

The usefulness of the acid-clearing test (ACT) was further evaluated in 21 normal subjects by investigating the effects of differing the body position, swallowing water, smoking, and drinking alcohol on the test results and reproducibility. The ACT was carried out according to a reported procedure, with some modifications. The pH-electrode, which was calibrated against buffer solutions of pH 4.0 and 7.0 before each subject was investigated, was placed 5 cm above the lower esophageal sphincter (LES). The electrode was connected to a pH-meter. Fifteen milliliters of 0.1 M HCl was instilled in the esophagus over 30 sec, with the catheter placed 15 cm above the LES. The subject swallowed dry every 30 sec, and the number of swallows to reach a pH of 5.0 were summed. There was a significant increase in the number of swallows required to reach pH 5.0 in the head down position ($p<0.003$), after smoking ($p<0.003$), and after drinking alcohol ($p<0.001$). The number of swallows required to reach pH 5.0 was significantly lower in subjects who swallowed 5 ml of water ($p<0.0005$) than in those who swallowed dry. Five hundred and twenty-five milliliters of saliva or 1,680 ml of water was used to raise the pH of a 15-ml solution of 0.1 M HCl to 5.0. If saliva production during 15 min was drawn off every 30 sec, which corresponds to the amount swallowed during the same period in a normal subject, and this amount was mixed with the 0.1 M HCl solution, no change in pH occurred. It is proposed that the outcome of the ACT reflects both the transporting capacity of the esophageal muscles and the production of saliva. The precision of this test is unsatisfactory for use in individual cases, but it seems well suited for the evaluation of esophageal acid clearing in larger samples.

8132 RADIOIMMUNOASSAY OF GASTROINTESTINAL HORMONES. (Eng.) Straus, E. (Veterans Admin. Hosp., 130 W. Kingsbridge Rd., Bronx, NY 10468). *Gastroenterology* 74(1):141-152; 1978.

8133 LUMINAL GASTRIN STIMULATES GROWTH OF DISTAL RAT INTESTINE [Abstract]. (Eng.) Johnson, L. R.; Ryan, G. P.; Copeland, E. M.; Dudrick, S. J. (Univ. Texas Medical Sch., Houston, TX 77025). *Fed. Proc.* 37(3):374; 1978.

8134 THE EFFECT OF RENAL ARTERY LIGATION ON SERUM GASTRIN CONCENTRATIONS IN RATS [Abstract]. (Eng.) Wilder, D. E.; Hohnke, L. A. (Pfizer General Res., Groton, CT 06340). *Fed. Proc.* 37(3):373; 1978.

8135 EFFECTS OF INTESTINAL INFUSION OF SOLUTIONS WITH DIFFERENT TONICITIES IN CONTROL AND STAPHYLOCOCCAL ENTEROTOXIN B (SEB)-EXPOSED RABBITS [Abstract]. (Eng.) Dufault, D. R.; Liu, C. T. (USA Medical Res. Inst. Infectious Diseases, Fort Detrick, MD 21701). *Fed. Proc.* 37(3):697; 1978.

8136 MUCIN DEGRADATION IN THE BLIND LOOP SYNDROME [Abstract]. (Eng.) Prizont, R. (Albuquerque Veterans Admin. Hosp., Albuquerque, NM). *Clin. Res.* 26(2):112; 1978.

8137 A STUDY OF THE EFFICACY OF TYPHOID VACCINE IN INDUCING HUMORAL AND CELL-MEDIATED IMMUNE RESPONSES IN HUMAN VOLUNTEERS. (Eng.) Nath, T. R.; Malaviya, A. N.; Kumar, R.; Balakrishnan, K.; Singh, B. P. (All India Inst. Medical Sciences, New Delhi, 110016, India). *Clin. Exp. Immunol.* 30(1):38-43; 1977.

8138 MICROBIAL ECOLOGY OF THE GASTROINTESTINAL TRACT. (Eng.) Savage, D. C. (Dept. Microbiology, Univ. Illinois, Urbana, IL 61801). *Annu. Rev. Microbiol.* 31:107-133; 1977.

8139 FURTHER EVIDENCE FOR THE EARLY EXPRESSION OF IMMUNITY TO Fasciola hepatica IN RATS. (Eng.) Hayes, T. J. (Hoffmann-La Roche, Inc., Nutley, NJ 07110). *J. Parasitol.* 64(2):374-376; 1978.

See also, 7996, 8023, 8031, 8034, 8152.

DIAGNOSTIC PROCEDURES

8140 CURRENT STATUS OF CARCINOEMBRYONIC ANTIGEN (CEA) AND CEA-S ASSAYS IN THE EVALUATION OF NEOPLASMS OF THE GASTROINTESTINAL TRACT. (Eng.) Nakamura, R. M.; Plow, E. F.; Edgington, T. S. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037). *Ann. Clin. Lab. Med.* 8(1):4-10; 1978.

The significance of carcinoembryonic antigen (CEA) determination in the diagnosis of gastrointestinal neoplasms was compared with that of CEA-S (a homogeneous isometric species of CEA) in 308 sera. In addition, the properties of CEA and CEA-S and the results of several studies on CEA in the diagnosis of neoplasms are reviewed. The sedimentation velocity for CEA is slightly higher than that for CEA-S (6.8 versus 6.6). The isoelectric distribution of CEA is heterogeneous and extends from less than 3.0 to approximately 5.2 as compared with the homogeneous isoelectric point of approximately 4.5 for CEA-S. The amino acid composition of CEA and CEA-S is similar, except that CEA-S has a 10% relative increase in lysine and a 5% decrease in threonine content. In the study of the 308 sera, a threshold of 5 ng/ml was used for the CEA assay, and a threshold of 14 U/ml was used for the CEA-S assay. Four percent of the 75 normal sera and 13.6% of the sera from 44 patients with benign pulmonary diseases were positive for CEA, but none were positive for CEA-S. Similarly, the CEA test was positive in 9.8% of the sera from 92 patients with benign gastrointestinal disease and in 31.2% of the 48 patients with lung carcinoma, whereas the corresponding figures for the CEA-S assay were 0%, and 10.4%, resp. The CEA assay was positive in 34.7% of the 49 cases of gastrointestinal tumors, whereas the CEA-S assay was positive in 46%. These results suggest that the CEA-S assay results in fewer false-positive results and in more true-positive results in gastrointestinal malignancies than does the conventional CEA assay.

8141 ANGIOGRAPHIC DIAGNOSIS OF LEIOMYOMAS OF THE SMALL INTESTINE. (Eng.) Han, S. Y.; Aldrete, J. S. (Univ. Alabama Sch. Medicine, 619 S. 19th St., Birmingham, AL 35233). *Am. J. Gastroenterol.* 68(1):91-94; 1977.

The case of a 54-yr-old man with recurrent episodes of severe gastrointestinal bleeding over a 5-yr period in whom small intestinal leiomyomas were demonstrated by visceral angiography is presented. The patient had experienced several episodes of unexplained hematochezia. Endoscopy of the esophagus, stomach, and duodenum, and proctosigmoidoscopy were unrevealing. Mesenteric arteriography demonstrated three vascular lesions in the small bowel. The largest measured 8 cm in diameter and was located in the distal ileum, and the other two were found in the proximal jejunum. The arteries supplying these tumors were dilated and opacified earlier than the adjacent normal vessels. There was also early visualization of effluent veins that appeared markedly dilated. At operation, multiple sessile and pedunculated tumors were found arising from the muscular layers of the small intestine. Most could be shelled out without resection of the small bowel. Removal of the large tumor required resection of the involved ileal segment. Histological examination of all tumors showed leiomyomas with cystic degeneration, hemorrhage, and infarct but without evidence of sarcomatous changes. The results in this patient and those reported in the literature suggest that leiomyomas of the small intestine may be detected by their peculiar characteristics on selective arteriography, and that this procedure may be useful in patients with massive hematochezia of unexplained origin.

8142 THE PERSISTENT LOOP SIGN IN NEONATAL NECROTIZING ENTEROCOLITIS: A NEW INDICATION FOR SURGICAL INTERVENTION? (Eng.) Wexler, H. A. (2485 N.E. 202nd St., N. Miami Beach, FL 33180). *Radiology* 126(1):201-204; 1978.

Five patients who demonstrated a new radiographic sign in neonatal necrotizing enterocolitis are described. All patients demonstrated an unchanging dilated loop of small bowel, which was followed by perforation in three infants; the other two had surgical and/or autopsy corroboration of profound bowel wall necrosis. In the first child, early surgical intervention was not recommended because this was one of the first in which a "persistent loop" was recognized. Perforation occurred, and emergency laparotomy confirmed peritonitis with distal ileal and jejunal necrosis. Although the persistent loop syndrome was recognized in the second patient, her temporary clinical improvement delayed surgical intervention. By the time she was operated upon, she had advanced bowel necrosis, peritonitis, and ascites. In the third patient, it was not possible to resect all diseased bowel at laparotomy, and the unchanging bowel pattern in the first 3 postoperative days suggested that the remaining necrotic intestine was irreparably damaged. Since the clinical condition of the fourth child did not warrant an emergency laparotomy after an initial radiograph showed the persistent loop syndrome, one more radiograph was obtained; by this time, perforation had already occurred. The morbid condition of the fifth patient precluded surgical intervention at the time that the persistent loop was identified. Loculated free air in the right lower quadrant was noted on the next film. At autopsy, the perforation had closed over; however, as in the other cases, it was preceded by a persistent loop. These findings suggest that infants with a persistent loop be considered candidates for laparotomy.

8143 CHOLANGIOGRAPHIC ABNORMALITIES IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE. (Eng.) Rohrmann, C. A., Jr.; Ansel, H. J.; Freeny, P. C.; Silverstein, F. E.; Protell, R. L.; Fenster, L. F.; *et al.* (Univ. Washington Sch. Medicine, Seattle, WA). *Radiology* 127(3):635-641; 1978.

An analysis of cholangiographic abnormalities present in 20 patients with inflammatory bowel disease (IBD) who underwent endoscopic retrograde cholangio-

graphy is presented. In all patients, abnormal liver function tests suggested major duct obstruction. The extent of involvement of both intra- and extrahepatic systems, branching characteristics, and caliber abnormalities manifested by mural irregularities, nodules, diverticula, stenoses, ectasias, and ductal obstructions or obliterations were evaluated by cholangiography. Intrahepatic system abnormalities were found in all patients. They included diminished arborization or pruning in 16 patients, stenosis and ectasia in 15 patients, and severe obliterative changes with duct obstruction in 4 patients. Extrahepatic duct abnormalities observed in 15 patients included mural irregularity, marked in some cases, diverticula, and stenoses. The cystic duct or cystic duct remnant was abnormal in six of eight patients who had previously undergone cholecystectomy. An extrahepatic ductal abnormality apparently found exclusively in IBD patients was a saccular outpouching of the lumen producing a diverticular appearance. The characteristic cholangiographic abnormalities demonstrated make differential diagnosis of biliary tract diseases possible. Endoscopic retrograde cholangiography is recommended to exclude extrahepatic obstruction, to establish a nonoperative diagnosis, and to assist in determining the method of treatment.

8144 STUDIES ON ALKALINE PHOSPHATASE ISOENZYMES IN HEPATIC DISEASES, RELATION TO γ-GLUTAMYLTRANSFERASE. (Eng.) Burlina, A.; Bugiardini, R. (Centro Ospedaliero Borgo Trento, 37100 Verona, Italy). *Clin. Chim. Acta* 85(1):49-54; 1978.

Activities of the isoenzymes of alkaline phosphatase (ALP) and of total γ-glutamyltransferase (γGT) were determined in the sera of 164 patients with hepatic diseases. Total ALP activity was increased over that in 40 normal controls in 45% of the patients with acute viral hepatitis, in 42% of the patients with chronic active hepatitis, in 47% of those with liver cirrhosis, and in all patients with biliary obstruction. Of the 86 patients with increased total ALP levels, 37% showed a wider ALP_2 band (the fraction migrating with α_2-globulin), 44% showed an ALP_1 band (biliary band), and 18% showed the ALP_4 band (cirrhotic band). The ALP_1 band was present in all cases of biliary obstruction, in 9% of cases of acute viral hepatitis, in 8% of chronic hepatitis cases, and in 14% of cases of cirrhosis. In the patients with a wider ALP_2 band, the γGT levels were increased but always lower than 200 U/l. In the patients with the ALP_1 band, the γGT values were all high, and 64% of the patients had a level higher than 200 U/l. In the patients with an ALP_4 band, the values were all lower than 200 U/l. There was a significant correlation between the presence of the ALP_1 fraction and increased γGT values (r=0.733). It appears that alkaline phosphatase fractionation gives diagnostic information not obtainable from the determination of total ALP and γGT levels.

8145 PERCUTANEOUS BIOPSY OF THE LIVER AS A PROCEDURE FOR OUTPATIENTS. (Eng.) Knauer, C. M. (Santa Clara Valley Medical Center, 751 S. Bascom Ave., San Jose, CA 95128). *Gastroenterology* 74(1):101-102; 1978.

Experience with percutaneous liver biopsy as an outpatient procedure in 107 patients during an 18-month period is presented. The Klatskin needle with the suction technique of Menghini is used, usually via the intercostal approach. The outpatient is allowed to leave 6 hr after the biopsy and is instructed to avoid athletic activity and heavy lifting for the next 24 hr. The outpatients ranged in age from 8 to 73 yr. Only one patient had a complication; he bled from the biopsy site within 1 hr of the procedure, requiring hospitalization for blood transfusion and surgical control of the bleeding site. The minimal cost saved per patient was $153.00. Liver biopsy in outpatients is both safe and economical.

8146 DIAGNOSIS OF HEPATOBILIARY DISEASE BY ^{99m}Tc-HIDA CHOLESCINTIGRAPHY. (Eng.) Rosenthall, L.; Shaffer, E. A.; Lisbona, R.; Pare, P. (Montreal General Hosp., 1650 Cedar Ave., Montreal, Quebec H3G 1A4, Canada). *Radiology* 126(2): 467-474; 1978.

The use of ^{99m}technetium-labeled N-substituted iminodiacetic acid derivative (Tc-HIDA) cholescintigraphy for the diagnosis of hepatobiliary disease was evaluated in 18 patients with acute or chronic cholecystitis, 17 patients with hepatocellular disease, 16 patients with biliary tract obstruction, and 19 controls with no evidence of hepatobiliary disease. In controls, images of the biliary ducts and gallbladder were obtained within 30 min after an i.v. injection of Tc-HIDA, while excretion from the intestinal tract was observed less than 60 min after injection. None of the patients with acute or chronic cholecystitis and cystic duct obstruction demonstrated gallbladder filling. In chronic cholecystitis with a patent cystic duct, the gallbladder was not visualized with Tc-HIDA scanning in 7 of 12 cases. This failure was rectified in one patient by administering cholecystokinin (CCK) followed by a second dose of Tc-HIDA. In seven patients who had surgical bypass procedures, obstruction as the cause of juandice was excluded because of gut activity within 60 min, no unusual dilatation of the biliary ducts beyond what is normally observed postoperatively, and a lack of prolonged retention of radioactivity in the biliary system. All five patients with serum bilirubin levels exceeding 16 mg% who were studied on consecutive days with Tc-HIDA and then ^{131}I-labeled rose bengal showed higher liver-to-background ratios with the latter agent; however, no gut excretion was appreciated with either radiopharmaceutical. Accurate visualization of the ductal system was possible with Tc-HIDA in 11 of 12 patients with hepatocellular disease in whom the serum bilirubin was between 0.8 and 4.6 mg%. No ductal visualization occurred with serum bilirubin levels above 5 mg%. In eight patients with partial extrahepatic obstruction and hyperbilirubinemia up to 5 mg%, a dilated biliary tree was detected by increased radioactivity, the diameter

of the ducts, and delayed washout of the test agent into the duodenum. In summary, Tc-HIDA cholescintigraphy can identify patients with acute and chronic cholecystitis and all patients with a patent cystic duct following CCK administration. The value of Tc-HIDA over standard radiography in jaundiced patients is demonstrated by its simplicity in making a definitive differential diagnosis possible when the serum bilirubin levels are 5 mg% or less.

8147 VALUE OF THE MANDIBULAR RADIOGRAPH IN FAMILIAL POLYPOSIS COLI. (Eng.) Rabin, M. S.; Price, H. I. (Johannesburg Hosp., Johannesburg, South Africa). *S. Afr. Med. J.* 53(12):457-459; 1978.

8148 FIBEROPTIC PANSIGMOIDOSCOPY: AN EVALUATION OF ITS USE IN AN OFFICE PRACTICE. (Eng.) Manier, J. W. (Marshfield Medical Foundation, Inc., 510 North St. Joseph's Ave., Marshfield, WI 54449). *Gastrointest. Endosc.* 24(3):119-120; 1978.

8149 THE VALUE OF DIAGNOSTIC AIDS IN DETECTING PANCREAS CANCER. (Eng.) Fitzgerald, P. J.; Fortner, J. G.; Watson, R. C.; Schwartz, M. K.; Sherlock, P.; Benua, R. S.; *et al.* (Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021). *Cancer* 41(3):868-879; 1978.

8150 ROUTINE OFFICE USE OF THE 60-CM FLEXIBLE FIBEROPTIC COLONOSCOPE. (Eng.) Carter, H. G. (Baylor Medical Plaza, 707 Barnett Tower, 3600 Gaston Ave., Dallas, TX 75246). *Dis. Colon Rectum* 21(2):101-103; 1978.

8151 PRELIMINARY ABDOMINAL FILMS IN ORAL CHOLECYSTOGRAPHY: ARE THEY NECESSARY? (Eng.) Harned, R. K.; LeVeen, R. F. (Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB 68105). *Am. J. Roentgenol.* 130(3):477-479; 1978.

8152 DISCRIMINATION OF HEPATOBILIARY DISEASES BY THE EVALUATION OF BROMSULFOPHTHALEIN BLOOD KINETICS. (Eng.) Molino, G.; Milanese, M.; Villa, A.; Cavanna, A.; Gaidano, G. P. (Istituto di Medicina Interna, Universita di Torino, Via Genova, 3, 10126 Torino, Italy). *J. Lab. Clin. Med.* 91(3):396-408; 1978.

8153 SALIVARY GLAND SCINTIGRAPHY: DIAGNOSTIC VERIFICATION OF PAROTID SALIVA FLOW THROUGH THE MAXILLARY SINUS AND NOSE. (Eng.) Karlsson, G.; Lind, M. (Karolinska sjukhuset, S-10401 Stockholm 60, Sweden). *Arch. Otorhinolaryngol.* 218(3/4):263-267; 1978.

8154 DIAGNOSIS OF PANCREATIC DISEASES. (Eng.) Lopez-Majano, V.; Bowie, J. (Cook County Hosp., 1825 W. Harrison St., Chicago, IL 60612). *Int. Surg.* 63(1):48-53; 1978.

8155 COMPARISON OF THE PRACTICAL DIAGNOSTIC VALUE OF DIFFERENT TOMOGRAPHIC MOVEMENTS. (Eng.) Labde, S.; Vuoria, P. (Univ. Central Hosp., SF 90220 Oulu 22, Finland). *Roentgenblaetter* 30(12):636-641; 1977.

8156 OSMOTIC EFFECT AND SOLUBILITY OF AMIPAQUE (METRIZAMIDE) IN THE GASTROINTESTINAL TRACT. (Eng.) Johansen, J. G.; Kolmannskog, S. (Tromso Regional Hosp., 9012 Tromso, Norway). *Invest. Radiol.* 13(1):93-97; 1978.

8157 A DIAGNOSTIC APPROACH TO INFLAMMATORY DISEASE OF THE PANCREAS BY MEANS OF ENDOSCOPIC RETROGRADE CHOLANGIO-PANCREATOGRAPHY. (Eng.) Harada, H.; Tanaka, J.; Shundo, T.; Hayashi, T.; Sasaki, T.; Yamamoto, N.; *et al.* (Okayama Univ. Medical Sch., 5-1 Shikada-cho, 2-chome, Okayama 700, Japan). *Gastroenterol. Jpn.* 12(5):387-394; 1977.

8158 COMPLICATIONS OF ENDOSCOPIC PAPILLOTOMY [Abstract]. (Eng.) Tytgat, G. (No affiliation given). *Scand. J. Gastroenterol.* 12(Suppl. 47):45; 1977.

8159 ENDOSCOPIC RETROGRADE CHOLANGIO-PANCREATOGRAPHY (ERCP): PRESENT POSITION AND PAPILLOTOMY. (Eng.) Siegel, J. H. (New York Medical Coll., Fifth Ave. at 106th St., New York, NY 10029). *N.Y. J. Med.* 78(4):583-591; 1978.

8160 ANGIOGRAPHY IN DETERMINING THE CAUSE AND TREATMENT OF GASTROINTESTINAL BLEEDING. (Eng.) Grace, D. M.; Gold, R. E. (Univ. Hosp., 339 Windermere Rd., London, Ontario N6A 5A5, Canada). *Can. J. Surg.* 21(2):171-174; 1978.

8161 JEJUNAL BIOPSY WITHOUT THE NEED FOR SCREENING. (Eng.) Holdstock, G. (Royal Victoria Hosp., Boscombe, Bournemouth, England). *Lancet* 1(8076):1236-1237; 1978.

8162 DIAGNOSIS OF GASTRO-OESOPHAGEAL REFLUX IN INFANTS AND CHILDREN BY METHYLENE-BLUE TEST. (Eng.) Girardi, G.; Fritis, E.; Vial, L.; Hevia, E.; Otero, M.; Larrain, A.; Pope, C. E., II (E. Gonzalez Cortes Hosp., Santiago, Chile). *Lancet* 1(8076):1236; 1978.

8163 ROENTGEN EXAMINATION OF THE SMALL BOWEL USING INTRALUMINAL CONTRAST MEDIA. (Eng.) Dodds, W. J.; Goldberg, H. I. (Milwaukee County Medical Complex, 8700 W. Wisconsin Ave., Milwaukee, WI 53226). *Am. J. Dig. Dis.* 23(6):550-552; 1978.

8164 COST OF CARRYING OUT CLINICAL DIAGNOSTIC TESTS. (Eng.) Barnard, D. J.; Bingle, J. P.; Garratt, C. J. (York District Hosp., York YO3 7HE, England). *Br. Med. J.* 1(6125):1463-1465; 1978.

8165 STUDIES ON THE CEA-LIKE SUBSTANCE IN GASTRIC JUICE. (Eng.) Kawaharada, M.; Fukuya, S.; Suzuki, T.; Sudo, M.; Akahonai, Y.; Yachi, A.; Wada, T. (Sapporo Medical Coll., S. 1, W. 16, Chuo-ku, Sapporo, 060 Japan). *Gastroenterol. Jpn.* 13(1):8-12; 1978.

8166 COLONIC ENDOSCOPY IN PERSPECTIVE. (Eng.) Talbott, T. M.; MacKeigan, J. M. (Michigan State Coll. Human Medicine, Grand Rapids, MI 49503). *Surg. Clin. North Am.* 58(3):459-468; 1978.

8167 CLINICAL VALUE OF GASTROINTESTINAL FIBER-OPTIC ENDOSCOPY. (Eng.) Groll, A. (Div. Gastroenterology, Queen's Univ., 102 Stuart St., Kingston, Ontario K7L 2V6, Canada). *Can. J. Surg.* 21(3):196-205; 1978.

8168 TESTS OF SMALL-INTESTINAL FUNCTION--DIGESTION, ABSORPTION, SECRETION. (Eng.) Russell, R. I.; Lee, F. D. (Royal Infirmary, Glasgow, Scotland). *Clin. Gastroenterol.* 7(2):277-315; 1978.

8169 INVESTIGATION OF THE GASTROINTESTINAL BACTERIAL FLORA. (Eng.) Borriello, P.; Hudson, M.; Hill, M. (Central Public Health Lab., London, England). *Clin. Gastroenterol.* 7(2):329-349; 1978.

8170 BREATH TESTS. (Eng.) Caspary, W. F. (Dept. Medicine, Univ. Gottingen, Gottingen, W. Germany). *Clin. Gastroenterol.* 7(2):7(2):351-374; 1978.

8171 ENDOSCOPIC CYTOLOGY AND BIOPSY IN THE UPPER GASTROINTESTINAL TRACT. (Eng.) Hughes, H. E.; Lee, F. D.; Mackenzie, J. F. (Dept. Cytology, Univ. Glasgow, Glasgow, Scotland). *Clin. Gastroenterol.* 7(2):375-396; 1978.

8172 DOUBLE CONTRAST RADIOLOGY IN THE GASTRO-INTESTINAL TRACT. (Eng.) James, W. B. (Southern General Hosp., Glasgow, Scotland). *Clin. Gastroenterol.* 7(2):397-430; 1978.

8173 GASTROINTESTINAL ANGIOGRAPHY. (Eng.) Boijsen, E.; Tylen, U. (Univ. Hosp., Lund, Sweden). *Clin. Gastroenterol.* 7(2):431-452; 1978.

8174 VISUALIZATION OF THE LIVER, BILIARY TREE AND PANCREAS. PART I: RADIOLOGY. (Eng.) Nunnerley, H. B. (King's Coll. Hosp., London, England). *Clin. Gastroenterol.* 7(2):453-475; 1978.

8175 DIFFERENTIAL DIAGNOSIS OF UPPER GASTRO-INTESTINAL BLEEDING AND CANCER. (Eng.) Sherlock, P.; Winawer, S. J. (Memorial Sloan-Kettering Cancer Center, New York, NY). *CA* 28(1):7-16; 1978.

8176 BARIUM DUODENOGRAPHY AS AN ADJUNCT TO PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY. (Eng.) Jaques, P. F.; Bream, C. A. (Dept. Radiology, Univ. North Carolina, Chapel Hill, NC 27514). *Am. J. Roentgenol.* 130(4):693-696; 1978.

8177 ULTRASOUND IN THE INVESTIGATION OF GALL-BLADDER DISEASE. (Eng.) Kappelman, N. B.; Sanders, R. C. (Johns Hopkins Medical Inst., 601 N. Broadway, Baltimore, MD 21205). *JAMA* 239 (14):1426-1428; 1978.

See also, 8013, 8131, 8132, 8200, 8204, 8205, 8214, 8220, 8222, 8243, 8251, 8252, 8263, 8267, 8277, 8285, 8286, 8295, 8296, 8315, 8320, 8344, 8345, 8346, 8358, 8369, 8371, 8376, 8377, 8381, 8396, 8399, 8400, 8401, 8402, 8407, 8411, 8412, 8427, 8438, 8446, 8447, 8452, 8459, 8460, 8463, 8464, 8465, 8466, 8467, 8472, 8473, 8474, 8476, 8494, 8517, 8536, 8537, 8561, 8563, 8577, 8578, 8598, 8608.

GASTROINTESTINAL DISEASES

ESOPHAGUS

8178 RECURRENT BRONCHOPNEUMOPATHIES CAUSED BY GASTROESOPHAGEAL REFLUX IN THE CHILD. CLINICAL, RADIOLOGICAL, AND HISTOLOGICAL STUDY OF 36 CASES. (Fre.) Dutau, G.; Familiades, J.; Rochioccioli, P. (C.H.U. Rangueil, 31054 Toulouse Cedex, France). *Poumon Coeur* 33(6):351-359; 1977.

In the period January 1973-March 1977, 36 children (ages 3 months-15 yr) who had recurrent bronchitis associated with gastroesophageal reflux (GF) were studied. The bronchitis had to have occurred at least three times in 3 months, and it was often associated with a chronic cough. X-ray studies were performed with the patients in the orthostatic, procubitus, and decubitus dorsal position with Trendelenburg. Respiratory endoscopy was performed in 5 patients and esophageal endoscopy in 14. The x-ray studies did not show any pulmonary abnormalities in 22% of the children; uni- or bilateral paracardiac opacities were observed in 36%, diffuse opacities with tendency for retraction of the right upper lobe in 8%, parenchymal distention in 22%, obstructive emphysema in 5%, and bronchial inundation in 8%. Pulmonary lesions were observed in all five of the children who were examined endoscopically. The lesions observed were either localized with atelectasis, obstructive emphysema, or bronchiectasis or generalized with granulomatous reactions around food particles. The GF problems were caused by neonatal vomiting in 34%, actual vomiting in 56%, postural vomiting in 28%, and nocturnal respiratory distress associated with vomiting in 16%. It is concluded that older children often have respiratory signs of GF before gastrointestinal signs, and GF may be present for several months or years before diagnosis.

8179 SCLEROTHERAPY OF BLEEDING OESOPHAGEAL VARICES BY MEANS OF ENDOSCOPY. (Eng.) Paquet, K. J.; Oberhammer, E. (Dept. Surgery, Univ. Bonn, D-5300 Bonn-Venusberg, W. Germany). *Endoscopy* 10(1):7-12; 1978.

The treatment of hemorrhage from gastroesophageal varices by endoscopic sclerotherapy of the esophageal wall is reported in 640 patients treated over a period of 8 yr. Using a special rigid esophagoscope, which included an optical system, an aspirator, and a needle and syringe, a maximum of 1 ml of 1% Aethoxysclerol (total fluid volume not more than 50 ml) was injected into the esophageal epithelium adjacent to the varices (if sclerosing agents are partly or totally injected into the esophageal varices, hemorrhage from varices of the stomach will occur). After two to four injections adjacent to the varices during 1 week, the remission of the varices was documented endoscopically or radiographically. Patients required reevaluation at 4-6 month intervals. After two to three sessions of sclerotherapy, the interval of control could be extended up to 1 yr without new danger of hemorrhage. If hemorrhage recurred or if increasing size of the varicosities was observed, sclerotherapy was repeated. If liver function improved, a portasystemic shunt was performed. In 211 patients, the indication for sclerotherapy was uncontrollable hemorrhage; in 386 patients it was performed in the bleeding-free interval for either decompensated liver function, hepatic precoma, the absence of anastomosable portal vessels, or thrombosed shunts. The remaining 43 patients were treated because of impending hemorrhage and portal hypertension. The overall mortality rate was 14.5%, the main causes of death being liver coma (42/93 patients), uncontrollable hemorrhage (26/93), and mediastinitis and pyothorax (8/93). About 50% of the patients survived for periods of 1-8 yr. Most of the 65% still alive have experienced no further bleeding. It is concluded that sclerotherapy is the method of choice in the treatment of massive hemorrhage from esophageal varices. It is indicated in patients with decompensated liver function and whenever a shunt procedure is impossible or inadvisable.

8180 ULCERATIVE ESOPHAGITIS AND COLITIS IN A PEDIATRIC PATIENT WITH BEHCET'S SYNDROME: RESPONSE TO STEROID THERAPY. (Eng.) Lebwohl, O.; Forde, K. A.; Berdon, W. E.; Morrison, S.; Challop, R. (Columbia Univ. Coll. Physicians and Surgeons, 630 W. 168th St., New York, NY 10032). *Am. J. Gastroenterol.* 68(6):550-555; 1977.

The case of a patient with Behcet's syndrome who developed radiologic and endoscopic evidence of ulcerative esophagitis and colitis is presented. The patient had been noted since infancy to have recurrent painless oral ulcers. At 11 yr of age, she developed painful genital and perianal ulcers, and oral ulcers recurred. Her symptoms resolved with treatment, which included prednisolone. Six months later, she presented with diffuse abdominal pain and persistent oral ulcers. Stool guaiac tests were positive, but sigmoidoscopy and barium enema examinations were normal. The symptoms resolved, and the patient did well on varying doses of prednisone for over 2 yr. At this time, she developed frontal headaches, episodes of nausea and vomiting, photophobia, diplopia, and oral ulcers, all attributed to Behcet's syndrome with central nervous system involvement. The symptoms gradually resolved while she continued to take 10 mg of prednisone every other day. One year later, she developed epigastric pain, and an upper gastrointestinal series revealed a large posterolateral ulcer in the lower esophagus, felt to be an impending perforation. Stool hematest was intermittently positive, and a barium enema examination showed polypoid filling defects in the cecum and ascending colon. The patient's symptoms gradually improved after the prednisone dose was increased to 60 mg per day. A repeat esophagram demonstrated partial healing of the esophageal ulcer. Her abdominal symptoms recurred after tapering the steroid dose. Fiberoptic colonoscopy revealed a localized area of severe inflammatory bowel disease characterized by ulceration, shaggy

fibrinous exudate, friable mucosa, mucosal pseudopolyps, and some narrowing of the lumen, all in the cecum and proximal colon. The patient's symptoms resolved after the steroid dose was again increased to 60 mg daily, and a repeat barium enema showed considerable improvement in the appearance of the cecum and ascending colon. Colitis and esophageal ulceration should be considered in patients with Behcet's syndrome who present with gastrointestinal symptoms.

8181 THE RADIOLOGICAL DIAGNOSIS OF PEPTIC ULCER OF THE ESOPHAGUS. (Ger.) Truber, E.; Treichel, J. (Klinikum Steglitz der Freien Universitat, Hindenburgdamm 30, 1000 Berlin A5, W. Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 128(2):111-115; 1978.

Peptic esophageal ulcer was diagnosed radiologically in 14 patients. The radiological examination also demonstrated esophagogastric reflux in all 14, esophagitis in 10, axial hiatal hernia in 13, and stenosis in 2. The ulcer was located at the esophagogastric junction in 13 cases and 5 cm above the cardia in the other. Endoscopy confirmed the x-ray diagnosis in all 11 cases examined. Hypotonic esophagography is believed essential for the differentiation between benign and malignant ulcers.

8182 BIOTIN-RESPONSIVE PROPIONIC ACIDEMIA PRESENTING AS THE RUMINATION SYNDROME. (Eng.) Hillman, R. E.; Keating, J. P.; Williams, J. C. (St. Louis Children's Hosp., 500 S. Kingshighway Blvd., P.O. Box 14871, St. Louis, MO 63178). *J. Pediatr.* 92(3):439-441; 1978.

8183 HERPES SIMPLEX ULCERATIVE ESOPHAGITIS IN A HEALTHY SUBJECT. (Eng.) Depew, W. T.; Prentice, R. S. A.; Beck, I. T.; Blakeman, J. M.; DaCosta, L. R. (Dept. Gastroenterology, Univ. Southern California, Los Angeles, CA 90007). *Am. J. Gastroenterol.* 68(4):381-385; 1977.

8184 SQUAMOUS PAPILLOMA OF THE ESOPHAGUS. (Eng.) Dourdourekas, D.; Kaymakcalan, H.; Steigmann, F. (Cook County Hosp., 1835 W. Harrison St., Chicago, IL 60612). *Am. J. Gastroenterol.* 68(4):379-380; 1977.

8185 STUDIES ON EXOGENOUS AND ENDOGENOUS INTERACTION OF GASTRIN AND SECRETIN IN A CASE OF ACHALASIA. (Eng.) Sekiyama, N.; Kakumoto, Y.; Nakagawa, S.; Wada, T. (Sapporo Medical Coll., S-1, W-16, Chuoh-ku, Sapporo, 060 Japan). *Gastroenterol. Jpn.* 12(5):395-399; 1977.

8186 FOREIGN BODY IN THE OESOPHAGUS: REVIEW OF 2394 CASES. (Eng.) Nandi, P.; Ong, G. B. (Queen Mary Hosp., Hong Kong). *Br. J. Surg.* 65(1): 5-9; 1978.

8187 RATIONAL TREATMENT OF OESOPHAGITIS. (Eng.) de Carle, D. J. (St. George Hosp., Kogarah, New South Wales 2217, Australia). *Drugs* 15(3):231-234; 1978.

8188 A TRIPLE DUPLICATION OF THE ALIMENTARY TRACT. (Eng.) Sherman, N. J.; Morrow, D.; Asch, M. (Children's Hosp. of Los Angeles, Los Angeles, CA 90054). *J. Pediatr. Surg.* 13(2): 187-188; 1978.

8189 INTRAMURAL DIVERTICULOSIS OF THE ESOPHAGUS IN AN EIGHT-YEAR-OLD BOY. (Eng.) Braun, P.; Nussle, D.; Roy, C. C.; Cuendet, A. (Hopital Cantonal, CH-1211 Geneva 4, Switzerland). *Pediatr. Radiol.* 6(4):235-237; 1978.

8190 SQUAMOUS CELL PAPILLOMA OF THE ESOPHAGUS: REPORT OF A CASE AFTER PEPTIC ESOPHAGITIS AND REPEATED BOUGIENAGE WITH REVIEW OF THE LITERATURE. (Eng.) Parnell, S. A. C.; Peppercorn, M. A.; Antonioli, D. A.; Cohen, M. A.; Joffe, N. (Beth Israel Hosp., 330 Brookline Ave., Boston, MA 02215). *Gastroenterology* 74(5, Part 1):910-913; 1978.

8191 SURGICAL MANAGEMENT OF HIATAL HERNIA WITH ESOPHAGEAL REFLUX. (Eng.) Vansant, J. H. (Doctors' Clinic Center, Suite 105, 844 Kempsville Rd., Norfolk, VA 23502). *Am. Surg.* 44(4):179-195; 1978.

8192 THE MALLORY-WEISS SYNDROME: A PROSPECTIVE STUDY IN 130 PATIENTS. (Eng.) Clain, J. E.; Novis, B. H.; Barbezat, G. O.; Bank, S. (Groote Schuur Hosp., Observatory, Cape Town 7925, South Africa). *S. Afr. Med. J.* 53(15):596-597; 1978.

8193 GRANULAR CELL TUMORS OF THE ESOPHAGUS. (Eng.) Gershwind, M. E.; Chiat, H.; Addei, K. A.; Ferraro, L. R. (Nassau Hosp., Mineola, NY 11501). *Gastrointest. Radiol.* 2(4):327-330; 1978.

8194 ESOPHAGEAL STRICTURE SECONDARY TO CANDIDIASIS. (Eng.) Ott, D. J.; Gelfand, D. W. (North Carolina Baptist Hosp., 300 S. Hawthorne Rd., Winston-Salem, NC 27103). *Gastrointest. Radiol.* 2(4):323-325; 1978.

8195 TRAUMA TO THE ESOPHAGUS. (Eng.) Love, L.; Berkow, A. E. (Foster G. McGaw Hosp., 2160 S. First Ave., Maywood, IL 60153). *Gastrointest. Radiol.* 2(4):305-321; 1978.

8196 MID ESOPHAGEAL STRICTURE. (Eng.) Musher, D. R. (New York Univ. Sch. Medicine, New

York, NY). *Am. J. Gastroenterol.* 69(3, Part 1): 331-336; 1978.

8197 MALLORY-WEISS SYNDROME IN TWO CHILDREN. (Eng.) Lamiell, J. M.; Weyandt, T. B. (Tripler Army Medical Center, Honolulu, HI). *J. Pediatr.* 92(4):583-586; 1978.

See also, 8005, 8160, 8162, 8167, 8171, 8175, 8220, 8237, 8239, 8251, 8267, 8305, 8367, 8398, 8529, 8572, 8573, 8574.

STOMACH

8198 GASTRIC LESION AND PERNICIOUS ANEMIA: A FAMILY STUDY. (Eng.) Varis, K.; Stenman, U. H.; Lehtola, J.; Siurala, M. (Second Dept. Medicine, Univ. Helsinki, Helsinki, Finland). *Acta Hepatogastroenterol.* 25(1):62-67; 1978.

To investigate the genetic contribution in the deficient secretion of intrinsic factor (IF) that underlies pernicious anemia, 5 subjects with pernicious anemia, 17 of their first-degree relatives, 3 subjects with atrophic gastritis, and 9 of their first-degree relatives were studied gastroscopically, bioptically, functionally, and immunologically. The results showed similarities of morphology, function, and immunology of the stomach within each family studied. Relatives of subjects with pernicious anemia accumulated features generally seen in overt pernicious anemia: slight to severe atrophic gastritis with normal or slightly altered antral mucosa, achlorhydria, low secretion of IF, and high levels of serum gastrin and parietal call antibody. Two relatives actually had overt pernicious anemia as judged by IF secretion. It was possible to identify a subgroup of relatives with overt pernicious anemia or a high risk of pernicious anemia on the basis of elevated serum gastrin levels, achlorhydria, and low IF secretion. IF secretion below 500 U/hr was seen exclusively in association with IF antibodies in the gastric juice and advanced atrophy of the gastric body mucosa (except in one subject with a resection stomach); four of these nine subjects had overt pernicious anemia on the basis of decreased serum vitamin B_{12} levels. The family of a subject with atrophic gastritis showed high levels of parietal cell antibodies.

8199 EFFECT OF PROLONGED ADMINISTRATION OF ANTI-INFLAMMATORY AND ANALGESIC DRUGS ON GASTRIC MUCOSA. (Fre.) Pascal, J. P.; Tournut, J. J. (CHU Toulouse-Purpan, F 31052 Toulouse Cedex, France). *Gastroenterol. Clin. Biol.* 2(1):15-22; 1978.

A histological study of the gastric mucosa was undertaken in 43 patients who had been on anti-inflammatory and/or analgesic drugs for 1-30 yr because of various arthritis types (mainly rheumatoid arthritis). Principally, the drugs involved were ketoprofene, indomethacin, acetylsalycilic acid, corticoids, buprofen, and sodium diclofenate. A group of 25 patients hospitalized in the gastroenterological unit for functional disorders were studied for comparison. Esophagogastroduodenal endoscopy was performed on each patient, and 10 biopsies of the mucosa were taken along the gastric curvature. A repeat of the study was performed on 12 of the arthritis patients 5-8 months after interruption or decrease in dosage of their analgesic or anti-inflammatory medication. A single pathologist rated all the biopsies as superficial gastritis, superficial gastritis with atrophy, atrophy, intestinal metaplasia, or normal. Specimens rated as atrophic were three times more frequent in the group of patients on the anti-arthritic drugs. Distribution of gastric lesions was not different in the two groups of patients. Reexamination of the 12 patients who had stopped or decreased their medication showed a significant ($p<0.001$) decrease in the number of specimens rated as atrophic; the frequency of metaplasia had not changed in these patients. It is concluded that the drugs used by these patients cause atrophy of the gastric mucosa but not superficial gastritis or intestinal metaplasia.

8200 ^{67}Ga-CITRATE SCANNING IN GASTROINTESTINAL MALIGNANCIES. (Eng.) Douds, H. N.; Berens, S. V.; Long, R. F.; Caplan, G. E. (Memorial Medical Center, P.O. Box 23089, Savannah, GA 31403). *Clin. Nucl. Med.* 3(5):179-183; 1978.

The use of ^{67}Ga-citrate scanning in cases of gastrointestinal (GI) malignancy is reported in seven patients and surveyed in the literature to point out specific situations where gallium scanning may be diagnostic. Seven proven cases of GI malignancy in which initial ^{67}Ga scans were positive were collected. Cases 1 and 2 were adenocarcinomas of the stomach, case 3 was a mixed-cell histiocytic lymphoma involving the stomach and cervical nodes, case 4 was a gastric lymphoma, case 5 was a recurrent small bowel lymphoma at the ligament of Treitz, case 6 was a recurrent cecal adenocarcinoma, and case 7 was a recurrent rectal histiocytic lymphoma. GI x-ray studies were normal in cases 4, 5, and 6 and inconclusive in cases 1 and 2. In case 3, a positive GI series, showing thickening of gastric folds suspicious for lymphoma, was

supported by the ^{67}Ga scan, with abnormal gastric uptake from the fundus to the mid-antrum. In the normal or inconclusive GI series, increased gallium uptake during the ^{67}Ga scan indicated the location of the malignancy. In case 7, the recurrent rectal lymphoma was confirmed with the scan, which further revealed the presence of extensive upper abdominal disease. The literature is generally pessimistic regarding the value of gallium scanning in suspected GI malignancies, the major objection being its obscuring activity in bowel content. The authors emphasize that meticulous bowel cleansing prior to scanning and repeat scans at 72 and 96 hr improve the diagnostic yield and make it possible to visualize the abnormal uptake within the bowel itself. Increased use of ^{67}Ga scanning is recommended as a simple, noninvasive study that has been particularly useful in support of radiographic contrast studies of suspected carcinomas, in detecting the recurrence of known malignancy, in defining the extent of primary and metastatic disease, and as a diagnostic tool for debilitated patients.

8201 RELATIONSHIP BETWEEN DIET AND CARCINOMA OF THE STOMACH, COLON, RECTUM, AND PANCREAS IN FRANCE. (Fre.) Meyer, F. (Hopital E.-Herriot, Pavillon H, F 69374 Lyon Cedex 2, France). *Gastroenterol. Clin. Biol.* 1(12):971-982; 1977.

Age-standardized mortality rates for the years 1968 and 1969 for cancer of the stomach, colon, rectum, and pancreas were compared with the average consumption of bread, potatoes, fresh vegetables, fresh fruits, beef, eggs, fish, cheese, butter, oil, sugar, wine, and beer in eight districts of France. Consumption of foods that supply the same nutrients (butter/oil, wine/beer) showed significant ($p \leq 0.05$) negative correlations as did consumption of the pairs potatoes/fruits, butter/fruits, and butter/wine. There was a significant ($p \leq 0.05$) positive correlation in the consumption of bread/sugar/eggs, of oil/wine/vegetables, and of potatoes/beer (except in the North), fruits/oil, and fruits/vegetables. Beef consumption was not significantly correlated with the mortality rates for any of the cancers; moreover, the correlation was negative in sign for stomach cancer mortality (r=0.58) and colon cancer mortality (r=0.33). Because the food consumption patterns and cancer mortality patterns of the North and the Mediterranean regions are atypical of those in the other six regions, the sign, absolute value, and level of significance of the correlations varied considerably when first one and then both of these regions were excluded from the analysis. The significant ($p \leq 0.05$) correlations for all eight regions were between stomach cancer mortality and fruit (r=-0.66) and oil (r=-0.72) consumption; colon cancer mortality and potato (r=0.68), fruit (r=-0.83), butter (r=0.74), and oil (r=0.83) consumption; rectal cancer mortality and potato (r=0.82), fruit (r=-0.77), oil (r=-0.71), and beer (r=0.73) consumption; and pancreatic cancer mortality and potato (r=0.67), wine (r=0.69), and beer (r=0.73) consumption. When the North was excluded from the analysis, the most important differences in the correlations were that potato consumption lost its positive significance in colon, rectal, and pancreatic cancer mortality due to large decreases in the absolute value of r, butter consumption reached significance ($p \leq 0.05$) in correlation with stomach cancer (r=0.87), wine consumption lost its negative significance in correlation with pancreatic cancer, and beer consumption decreased below positive significance in correlation with rectal and pancreatic cancer. When the Mediterranean region was also excluded from the analysis, the most important changes were that potato consumption was again significant ($p \leq 0.05$) in stomach cancer mortality (r=0.86), the fruit consumption correlation changed markedly in either absolute value or sign and became insignificant in relation to colon and rectal cancer mortality, butter consumption became significant ($p \leq 0.05$) in rectal cancer mortality (r=0.90), and oil consumption lost its negative significance in colon cancer mortality. No testable hypothesis can yet be made from these data.

8202 BASIC EXPERIMENTS ON ORAL ADMINISTRATION OF 5-FLUOROURACIL EMULSION AS ADJUVANT CHEMOTHERAPY TO SURGICAL TREATMENT FOR GASTRIC CANCER. (Eng.) Watanabe, S.; Nakao, E.; Cho, K.; Nishioka, B.; Fujita, Y.; Takahashi, T.; *et al.* (Kyoto Prefectural Univ. Sch. Medicine, Kyoto, Japan). *Jpn. J. Surg.* 8(1):41-50; 1978.

The absorption and distribution of 5-fluorouracil (5-FU) into the blood and tissues of Donryu rats was investigated to evaluate the usefulness of 5-FU as adjuvant chemotherapy. An emulsion or solution of 5-FU (100 mg/kg body weight) was administered p.o. to the rats, and the 5-FU level was measured in the blood, kidneys, liver, stomach, duodenum, and lymph nodes at intervals of 30 min, 1, 2, 4, 6, 8, 12, and 24 hr. To investigate the absorption of 5-FU in the stomach in detail, the experiments were also performed on pylorus-ligated rats. The highest levels of 5-FU were found in the stomach and duodenum; the maximum concentration achieved by use of the emulsion was 1.5 times that after use of the solution. An appreciable concentration of 5-FU was maintained over a longer period of time by the emulsion. High levels of 5-FU were also detected in the lymph and lymph nodes. In the lymph, the maximum concentration attained by the 5-FU emulsion was three times higher than the maximum concentration attained by the 5-FU solution, and in the lymph nodes, the emulsion maximum concentration was twice as high as the solution maximum. The higher concentration was maintained over the course of the experiment. In pylorus-ligated rats, the 5-FU concentration in the mucosa of the stomach was 77.3 ± 38.7 μg/g following the 5-FU emulsion and 34.1 ± 17.3 μg/g following the solution. In the lymph nodes, the 5-FU concentration was 0.9 ± 0.31 μg/g for the emulsion and 0.4 ± 0.13 μg/g for the solution. The studies show that 5-FU can be absorbed directly from the mucous membrane of the stomach even when administered as an emulsion. An appreciable amount of 5-FU is transported to the lymph nodes by way of the lymphatic capillaries. The findings, in general, may be explained by the

enhancement of drug adsorbility, permeability, and retentivity in the tissues of the gastrointestinal tract when it is administered in the form of an emulsion. It is concluded that the p.o. administration of 5-FU emulsion is an effective adjuvant to surgery for gastric cancer, particularly for lymph node metastasis.

8203 TRANSPYLORIC EXTENSION OF GASTRIC CARCINOMA. (Eng.) Menuck, L. (Palomar Hosp., Escondido, CA 92025). *Am. J. Dig. Dis.* 23(3): 269-274; 1978.

The barium studies of the upper gastrointestinal tract in 88 patients with gastric carcinoma were evaluated to document the frequency of gross pyloric extension and to correlate the radiographic appearance with the gross anatomical changes in the pylorus and duodenal bulb. Of the 88 cases of gastric carcinoma, 17 showed transpyloric extension of the tumor; the phenomenon was microscopic in 10 cases and gross in 7. In all of the latter seven patients, the roentgen examination showed a malignant lesion of the antrum and/or pylorus with an associated abnormality of the duodenal bulb. The duodenal abnormalities included a mass lesion, mucosal destruction, or bizarre thickened folds. In six of the seven cases, the transpyloric extension was appreciated preoperatively. Three of the seven cases were properly diagnosed radiographically. Three cases were incorrectly diagnosed as gastric lymphoma because the examiner considered the finding of transpyloric extension to favor lymphoma, even though the antral abnormality was more consistent with carcinoma: The seventh case was initially believed to represent chronic peptic ulcer disease with antral and duodenal changes; the patient initially refused endoscopy, but the lesion was correctly diagnosed at 6-month follow-up. Only 3/88 cases showed pyloric obstruction. The present study fails to support the frequent assumption that since gastric carcinoma arises from the mucosa, it will encroach on the pylorus and obstruct it before transpyloric extension takes place.

8204 THE NATURAL HISTORY OF THE DOUBLE PYLORUS. (Eng.) Hegedus, V.; Poulsen, P. E.; Reichardt, J. (Glostrup Univ. Hosp., Copenhagen, DK-2600 Glostrup, Denmark). *Radiology* 126(1):29-34; 1978.

The natural history of the development of a double pylorus was investigated by examining the subjective complaints, clinical and radiological findings, and endoscopic observations in 11 patients with prepyloric ulcers and a fistula. Seven patients had undergone upper gastrointestinal series prior to the discovery of a fistula, and the prepyloric ulcers in these patients were located on the lesser curvature in four cases and the major curvature in two. Symptoms prior to the discovery of the antroduodenal fistula included abdominal pain, postprandial pain, vomiting, and gastrointestinal bleeding. In seven cases, significant relief of discomfort was obtained simultaneously with the establishment of the gastroduodenal fistula, and all these patients became asymptomatic. The remaining four patients were of doubtful clinical significance. One of these patients experienced recurrence of symptoms despite surgery. The consequences of the development of the fistula were difficult to evaluate in the other three patients because medical treatment was given at the time the double pylorus was discovered. Six case reports of patients in whom symptoms resolved after the development of the fistula are presented. One of these was particularly interesting, as the ulcer on the greater curvature did not affect the pancreas but penetrated into the duodenum. These cases demonstrate that the double pylorus is the result of ulcer penetration and that this fistula can be a sign of spontaneous recovery from ulcerative disease.

8205 DYNAMIC SCINTIPHOTOGRAPHIC STUDY OF THE ANTRAL MUCOSA WITH ^{99m}Tc-PERTECHNETATE IN HYPERGASTRINEMIA CAUSED BY RETAINED ANTRUM. (Ita.) Sciarretta, G.; Cavicchi, L.; Turba, E.; Fini, A.; Malaguti, P.; Cacciari, C. (Ospedali di Bologna, Bologna, Italy). *Bull. Sci. Med.* 149(2): 85-90; 1977.

The diagnosis of retained antrum by gastrin level test results and scintiphotographic studies is reported with reference to two male patients. Both patients had undergone Billroth II gastric resections for duodenal ulcers 6 and 9 months previously. The suspected diagnosis of retained antrum was based upon the elevated basal levels of serum gastrin in both of these patients (140 and 100 pg/ml compared with a normal value 60 ± 15 pg/ml). The tests of gastrin response (using secretin, calcium, and a protein meal) excluded the diagnosis of gastrinoma and supported the suspicion of retained antrum. In one of the patients, an endoscopic biopsy of the duodenal stump demonstrated the presence of antral mucosa. The scintiphotographic study with ^{99m}Tc-pertechnetate clearly demonstrated the presence of an area of activity on the bottom of the duodenal stump in both patients. The patients underwent resection of the retained antrum; histological examination of the tissue confirmed the diagnosis and follow-up studies revealed that the basal gastrinemia markedly decreased (to 75 pg/ml and 55 pg/ml resp.). The use of scintiphotography in cases of suspected retained antrum is advocated by the authors because it is a simple technique that gives a clear, differential diagnosis in these patients.

8206 A PROSPECTIVE COMPARATIVE STUDY OF CLINICAL AND HISTOLOGICAL CHARACTERISTICS IN ICELANDIC AND DANISH PATIENTS WITH GASTRIC ULCER, DUODENAL ULCER, AND X-RAY NEGATIVE DYSPEPSIA: I. DESIGN AND CLINICAL FEATURES. (Eng.) Oddsson, E.; Binder, V.; Thorgeirsson, T.; Jonasson, T. A.; Gunnlaugsson, O.; Wulff, M.; *et al.* (St. Josefspitallin, Reykjavik, Iceland). *Scand. J. Gastroenterol.* 12(6):689-694; 1977.

8207 NEW METHOD OF GASTRIC RESECTION. (Eng.) Cho, S.; Cho, S. (Yokohama Univ. Sch. Medicine, Yokohama, Japan). *Abdom. Surg.* 19(5): 107-111; 1977.

8208 EMPHYSEMATOUS GASTRITIS AND GASTRIC PERFORATION: AN UNUSUAL MANIFESTATION OF ALKALINE CORROSIVE INGESTION. (Eng.) Bernardino, M. E.; Lawson, T. L. (George Washington Univ. Medical Center, 901 23rd St. N.W., Washington, DC 20037). *Gastrointest. Radiol.* 2(2):107-108; 1977.

8209 SERUM GASTRIN IN PATIENTS WITH VARIOUS TYPES OF CHRONIC GASTRITIS. (Eng.) Fung, W. P.; Salmon, J. A.; Karim, S. M. M.; Tye, C. Y.; Lee, S. K. (Royal Perth Hosp., Box X2213, G.P.O. Perth, Western Australia 6001, Australia). *Am. J. Gastroenterol.* 68(2):141-144; 1977.

8210 ALCOHOL ABSORPTION FOLLOWING JEJUNAL INTERPOSITION IN PATIENTS WITH PREVIOUS GASTRIC SURGERY. (Eng.) Mendoza, C. B., Jr.; Rahbar, A.; Easley, G. W. (Veterans Admin. Hosp., Clarksburg, WV). *Milit. Med.* 142(11):874-875; 1977.

8211 MODIFIED HEINEKE-MIKULICZ PYLOROPLASTY AS AN EFFECTIVE GASTRIC DRAINAGE PROCEDURE. (Eng.) Seito, T.; Ogata, T. (Okayama Univ. Medical Sch., Okayama, Japan). *Jpn. J. Surg.* 7(3):165-169; 1977.

8212 UNUSUAL CAUSE OF GASTRIC BLEEDING: CRONKHITE-CANADA POLYPOSIS. (Eng.) Armengol-Miro, J. R.; Pedreira, J. D.; Guardia, J. (Ciudad Sanitaria de la Seguridad Social, Universidad Autonoma, Barcelona, Spain). *Endoscopy* 9(3): 177-179; 1977.

8213 GASTRIC CANCER IN THE HAWAII JAPANESE. (Eng.) Stemmermann, G. N. (Kuakini Medical Center, 347 N. Kuakini St., Honolulu, HI 96817). *Gann* 68(5):525-535; 1977.

8214 THE ULTRASONIC DEMONSTRATION OF GASTRIC LESIONS. (Eng.) Mascatello, V. J.; Carrera, G. F.; Telle, R. L.; Berger, M.; Holm, H. H.; Smith, E. H. (Georgetown Univ., Sch. Medicine, 3900 Reservoir Rd., N.W., Washington, DC 20007). *J. Clin. Ultrasound* 5(6):383-387; 1977.

8215 ISOLATED GASTRIC ANTRAL ULCERATION FROM INGESTION OF A SINGLE CLINITEST® TABLET. (Eng.) Colbert, P. M.; Sanders, P. D.; Frankl, H. (French Medical Clinic, Inc., 1941 Johnson Ave., San Luis Obispo, CA 93406). *Gastrointest. Endosc.* 24(2):82-83; 1977.

8216 SPLENIC VEIN THROMBOSIS: A CAUSE OF GASTRIC VARICES AND UPPER GASTROINTESTINAL BLEEDING. (Eng.) Amarnani, N. H.; Broor, S. L.; Varma, R. R.; Maddison, F.; Geenen, J. E. (Milwaukee County General Hosp., 8700 West Wisconsin Ave., Milwaukee, WI 53226). *Gastrointest. Endosc.* 24(2):83-85; 1977.

8217 IATROGENIC PROBLEMS FOLLOWING GASTRIC SURGERY. (Eng.) Jay, B. S.; Burrell, M. (Yale-New Haven Hosp., 333 Cedar St., New Haven, CT 06510). *Gastrointest. Radiol.* 2(3):239-257; 1977.

8218 POSTGASTROSTOMY DEFORMITY OF THE STOMACH. (Eng.) Bryk, D. (Jewish Hosp. and Medical Center of Brooklyn, 555 Prospect Place, Brooklyn, NY 11238). *Gastrointest. Radiol.* 2(3):259-261; 1977.

8219 SPLENOSIS SIMULATING AN INTRAMURAL GASTRIC MASS. (Eng.) Kutzen, B. M.; Levy, N. (Univ. Missouri Medical Center, Columbia, MO 65201). *Radiology* 126(1):45-46; 1977.

8220 pH-TESTED REFLUX WITHOUT HIATUS HERNIA. (Eng.) Lewicki, A. M.; Brooks, J. R.; Meguid, M.; Membreno, A.; Kia, D. (George Washington Univ. Medical Center, 901 23rd St. N.W., Washington, DC 20037). *Am. J. Roentgenol.* 130(1):43-45; 1978.

8221 FAMILIAL PYLORIC ATRESIA ASSOCIATED WITH EPIDERMOLYSIS BULLOSA. (Eng.) De Groot, W. G.; Postuma, R.; Hunter, A. G. W. (Health Sciences Children's Center, 685 Bannatyne Ave., Winnipeg, Manitoba, R3E OW1 Canada). *J. Pediatr.* 92 (3):429-431; 1978.

8222 DIFFERENTIATING GASTRIC TUMORS FROM BENIGN ULCERS. (Eng.) Bernardino, M. E.; Wyman, A. C.; Lawson, T. L. (George Washington Univ. Medical Center, 901 23rd St. N.W., Washington, DC 20037). *Va. Med.* 105(2):140-141; 1978.

8223 GLOMUS TUMOR OF THE STOMACH. (Eng.) Ichihashi, H.; Tonomura, K.; Kondo, T.; Nakashima, N. (Nagoya Univ. Sch. Medicine, Nagoya, Japan). *Jpn. J. Surg.* 7(4):253-257; 1977.

8224 GASTRIC AND DUODENAL PEPTIC ULCER DISEASE IN A WELL-DEFINED POPULATION: A PROSPECTIVE NECROPSY STUDY IN MALMO, SWEDEN. (Eng.) Lindstrom, C. G. (General Hosp., S-214 01 Malmo, Sweden). *Scand. J. Gastroenterol.* 13(2):139-143; 1978.

8225 PROPHYLAXIS WITH DEGLYCYRRHIZINISED LIQUORICE IN PATIENTS WITH HEALED

GASTRIC ULCER. (Eng.) Hollanders, D.; Green, G.; Woolf, I. L.; Boyes, B. E.; Wilson, R. Y.; Cowley, D. J.; Dymock, I. W. (Univ. Hosp. South Manchester, Manchester M20 8LR, England). *Br. Med. J.* 1(6106):148; 1978.

8226 MALAKOPLAKIA OF THE STOMACH: REPORT OF A CASE AND REVIEW OF THE LITERATURE. (Eng.) Nakabayashi, H.; Ito, T.; Izutsu, K.; Yatani, R.; Ishida, K. (Mie Univ. Sch. Medicine, 2-174 Endobashi, Tsu, Mie-Ken 514, Japan). *Arch. Pathol. Lab. Med.* 102(3):136-139; 1978.

8227 GANGRENE OF THE STOMACH: A CASE OF ACUTE NECROTIZING GASTRITIS. (Eng.) Strauss, R. J.; Friedman, M.; Platt, N.; Gassner, W.; Wise, L. (Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11040). *Am. J. Surg.* 135(2):253-257; 1978.

8228 HYPOGASTRINEMIA IN HYPOTHYROIDISM. (Eng.) Seino, Y.; Matsukura, S.; Inoue, Y.; Kadowaki, S.; Mori, K.; Imura, H. (Kobe Univ. Sch. Medicine, Kusunoki-cho, Ikuta-ku, Kobe, Japan). *Am. J. Dig. Dis.* 23(2):189-191; 1978.

8229 PERICARDIAL PERFORATION OF A GASTRIC ULCER SECONDARY TO PHYTOBEZOAR. (Eng.) Bianchi, C.; Di Bonito, L.; Fonda, F.; Sauli, G. (Ospedale Maggiore, Via Stuparich, 1, Trieste, Italy). *Panminerva Med.* 19(5):353-356; 1977.

8230 PRESENT AETIOPATHOGENETIC, PHYSIOPATHOLOGICAL AND PROPHYLACTIC QUESTIONS RELATING TO STRESS ULCER, WITH PARTICULAR REFERENCE TO VASCULAR SURGERY. (Eng.) Raso, A. M. (Second Sch. General Clinical Surgery and Surgical Therapy, Univ. Turin, Turin, Italy). *Panminerva Med.* 19(5): 307-314; 1977.

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8232 CELL PROLIFERATION, DIFFERENTIATION, AND MIGRATION IN THE GASTRIC MUCOSA: A STUDY ON THE BACKGROUND OF CARCINOGENESIS. (Eng.) Fujita, S.; Hattori, T. *In: Pathophysiology of Carcinogenesis in Digestive Organs. Proceedings of the 7th International Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo, 1976.* (Tokyo: University of Tokyo Press): 21-36; 1977.

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See also, 7974, 8005, 8030, 8031, 8037, 8038, 8039, 8041, 8042, 8043, 8044, 8046, 8048, 8049, 8050, 8051, 8052, 8140, 8160, 8162, 8165, 8167, 8169, 8171, 8175, 8178, 8182, 8191, 8192, 8197, 8259, 8266, 8267, 8271, 8272, 8273, 8280, 8281, 8285, 8287, 8292, 8398, 8402, 8437, 8439, 8507, 8566, 8572, 8573, 8574, 8595, 8596.

8257 IN-PATIENT TREATMENT OF PEPTIC ULCER WITH CIMETIDINE. I. EFFECT ON DUODENAL ULCER HEALING. (Ger.) Malchow, H.; Sewing, K. F.; Albinus, M.; Horn, B.; Schomerus, H.; Dolle, W. (Medizinische Universitatsklinik, Otfried-Muller-Strasse 10, 7400 Tubingen, W. Germany). *Dtsch. Med. Wochenschr.* 103(4):149-152; 1978.

The effectiveness of cimetidine in the treatment of peptic duodenal ulcer was studied in 67 patients in a 6-week double-blind trial. All patients received dimagnesium aluminum trisilicate (3 g/day). Thirty-three patients received cimetidine (4 x 300 mg/day p.o.) and 34 controls received placebo. Pain relief was significantly ($p<0.01$) better during the 1st week of treatment in the cimetidine-treated group than in the controls. Healing of the ulcers was observed in 16/33 patients in the cimetidine group and in 7/34 patients in the placebo group after the first 2 weeks of treatment, but there was no statistically significant difference at the end of the third week (23/33 versus 18/34, resp.), nor at the end of the 6-week treatment (30/33 versus 31/34, resp.). Neither basal nor pentagastrin-stimulated acid and pepsin secretions were affected by the 17-day administration of cimetidine. The drug had to be withdrawn in two cases because of elevated serum creatinine levels, but there were no other side-effects. Cimetidine had no long-term effects independent of the period of its presence in the blood. Compared with literature data on out-patients, the in-patient treatment caused faster healing in both the cimetidine and placebo groups.

8258 IN-PATIENT TREATMENT OF PEPTIC ULCER WITH CIMETIDINE. II. CONTROLLED DOUBLE-BLIND TRIAL IN GASTRIC ULCER PATIENTS. (Ger.) Sewing, K. F.; Malchow, H.; Albinus, M.; Horn, B.; Schomerus, H.; Dolle, W. (Pharmakologisches Institut der Universitat, Wilhelmstrasse 56, 7400 Tubingen, W. Germany). *Dtsch. Med. Wochenschr.* 103(4):152-154; 1978.

The effectiveness of cimetidine (4 x 300 mg/day p.o.) on the healing of peptic gastric ulcer was studied in 20 patients and in 16 placebo-treated controls in a double-blind trial. All patients received dimagnesium aluminum trisilicate (3 g/day). The average duration of the in-patient treatment was 28 days, and follow-up for healing continued for up to 6 weeks. The number of patients healed at the end of the 3rd week was 10/20 in the treated group versus 5/16 in the control group. At the end of the 6th week, the ulcers were healed in 17/20 patients versus in 10/16 placebo-treated controls; the difference was not statistically significant. There were also no significant differences in pain alleviation. Cimetidine had no influence on pentagastrin-stimulated acid and pepsin secretion. There were no serious untoward reactions to cimetidine.

8259 MAXIMUM ACID OUTPUT IN DUODENAL ULCER PATIENTS WITH DIFFERENT LENGTH OF HISTORY AND CONTROLS WITHOUT DYSPEPSIA. (Eng.) Kronberg, O.; Christiansen, P. M. (Bispebjerg & Hvidovre Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12 (8):957-962; 1977.

To investigate the hypothesis that maximum acid output (MAO) in duodenal ulcer (DU) patients is normal in those with an ulcer history of <3 yr but elevated in those with a longer history, MAO was measured in 3 groups of 21 men and 4 groups of 9 women with operatively verified DU and ulcer histories of <3, 3-6, or 7-10 yr. The results were compared with that of age-matched controls with no history of dyspepsia. An augmented histamine test was performed on patients and controls. The activity of the ulcer at surgery was evaluated as to the presence of an ulcer crater or duodenal scar. The rate of recurrent DU was studied in relation to the length of ulcer history. No significant relationship was found between the length of ulcer history and the activity of the ulcer or the recurrence rate. The risk of recurrent DU showed no tendency to increase with the length of history before vagotomy. For both men and women, the uncorrected mean values of MAO were significantly higher in patients with symptoms for <3 yr compared to controls (men, 42.1 ± 3.4 versus 24.2 ± 0.8 or 20.8 ± 1.6 mmol/hr in controls, $p<0.001$; women, 24.9 ± 2.9 versus 16.4 ± 1.5 mmol/hr in controls, $p<0.05$), but patients with longer histories had MAO values similar to those of the patients with <3 yr histories. When MAO values were corrected for weight or stature, they were significantly higher ($p<0.001$) than control values in all men (regardless of the length of ulcer history). Neither the men nor women showed any significant changes of MAO with increasing length of history. In a group of 11 men with duodenal ulcer histories of less than 1 yr, the MAO values (uncorrected and corrected) were also significantly higher ($p<0.001$) compared with controls. When groups of 16 men with histories of up to 22 yr were studied, a fixed proportion of hypersecretors (uncorrected MAO above 30 mmol/hr) was found among patients with up to an 18-yr ulcer history; when history exceeded 18 yr, a tendency toward smaller numbers of hypersecretors was noted. The present study does not support the hypothesis that the risk of ulcer recurrence after vagotomy and drainage is related to MAO before surgery, nor does it support the assumption that gastric hypersecretion is a result of duodenal ulceration. MAO was elevated above normal values in all ulcer patients, even those with histories of less than 1 or 3 yr.

8260 QUANTITATIVE STUDIES OF GASTRIN CELLS (G CELLS) AND PARIETAL CELLS IN RELATION TO GASTRIC ACID SECRETION IN PATIENTS WITH PEPTIC ULCER DISEASE. (Eng.) Stave, R.; Myren, J.; Brandtzaeg, P.; Gjone, E. (Rikshospitalet, Oslo, Norway). *Scand. J. Gastroenterol.* 13(3):293-298; 1978.

Stomach specimens from 42 patients subjected to partial gastric resection for gastric ulcer (12), duodenal ulcer and uremia (14), duodenal ulcer without uremia (11), and gastric cancer (5) were studied to determine possible relationships between acid output, parietal cell density in the body mucosa and the various parameters of the antral G cell population. Gastric acid secretion was measured before surgery. In the material as a whole, the antral G cell density was 35.6 ± 15.2, the G cell distribution was 71.7 ± 21.5 cm^2, the total antral G cell mass was 2,551 ± 1,780.3, and the parietal cell density in the body mucosa was 190.0 ± 89.4. The basal acid output (BAO) was 3.7 ± 4.3 mEq/hr and the pentagastrin-stimulated acid output (MAO) was 25.8 ± 15.7 mEq/hr. In the material as a whole there was a significant positive correlation between the parietal cell density and MAO ($r=0.589$, $p<0.01$), negative correlation between parietal cell density and age ($r=-0.365$, $p<0.02$), and positive correlation between antral G-cell mass and BAO ($r=0.331$, $p<0.05$). Analysis of the individual groups revealed significant correlations between parietal cell density and MAO in patients with duodenal ulcer and uremia ($r=0.711$, $p<0.05$) and in those with gastric cancer ($r=0.934$, $p<0.05$) and a borderline significance in the gastric ulcer patients ($r=0.520$). There was a significant negative correlation between parietal cell density and age in the group of patients with duodenal ulcer and uremia ($r=-0.606$, $p<0.05$). The correlation between G cell mass and BAO was at the borderline of significance in the gastric ulcer patients ($r=0.539$) and in the duodenal ulcer with uremia group ($r=0.537$). No correlation between the parietal cell density and the various parameters of the antral G cell population in the material as a whole or in any of the individual patient groups was found. There was a falling trend in gastric acid output with advancing age. Possible flaws in the experimental design, including the heterogeneity of the patient material as a whole, the limited categorization, the limited numbers of patients, and the inability to match patients with regard to age and sex are analyzed. The authors conclude that, as each of five hormones has the dual proper-smaller LA and LA:PY ratio increments; this did not occur with X. B usually increased LA and the LA:PY ratio. In the treatment of cirrhotic patients, a combination of G and insulin is recommended. If G cannot be used, then X or S is preferred to F. The simultaneous administration of sugars and insulin seems advantageous, while the combination of sugars with B appears risky.

8261 GASTRIC EMPTYING OF INGESTED ACID AND ITS EFFECTS ON PLASMA GASTRIN AND SECRETIN IN DUODENAL ULCER SUBJECTS. (Eng.) McLoughlin, J. C.; Green, W. E. R.; Buchanan, K. D. (Dept. Medicine and Surgery, Queen's Univ. Belfast, Belfast, Northern Ireland). *Scand. J. Gastroenterol.* 13(3): 313-319; 1978.

The gastric emptying of 250 ml of 0.1 M hydrochloric acid labeled with 1 mCi ^{113m}Indium chloride was followed in 16 normal and 15 duodenal ulcer (DU) subjects to evaluate their ability to handle a standard acid load. The time required for discharge of half of the load was significantly shorter in DU subjects than in controls (33.6 ± 2.7 versus 48.5 ± 5.3 min, $p<0.025$). The times required for the loss of the initial 20% of the load and for the intermediate phase of emptying were also significantly lower in the DU subjects than in controls (16.4 ± 2.5 versus 28.4 ± 4.8 min, $p<0.025$ and 20.8 ± 1.1 versus 27.4 ± 2.5 min, $p<0.0125$, resp.). In normal subjects, ingested acid suppressed plasma gastrin below basal values at 15 min ($p<0.025$), and significant suppression continued to the end of the study. In DU subjects, significant suppression ($p<0.05$) of plasma gastrin concentration occurred 45 min after acid ingestion, and suppression lasted to the end of the experiment. Controls showed no significant elevation in mean plasma secretin level at any point, but DU subjects demonstrated significant ($p<0.025$) elevation at 5 min after acid consumption. When the mean plasma secretin concentrations were calculated for the intermediate phase of gastric empyting (process at steady rate) the increment in plasma secretin showed significant correlation with the rate at which acid left the stomach (in DU subjects, $r=0.816$, $p<0.0025$ and in control, $r=0.688$, $p<0.01$). The point where the regression lines intersect the abscissa represents a quantitative acid threshold for measurable secretin release. It occurred in DU subjects at 0.413 mmol/min and in controls at 0.0298 mmol/min, $p<0.01$. The higher threshold in DU subjects suggests that defective secretin release at intermediate acid flow rates results in defective augmentation of the acid-neutralizing processes by secretin-stimulated pancreatic juice at high levels of acid production. The results show that DU subjects as a group discharge an acid load from the stomach more rapidly than do control subjects. The appearance of a quantitative threshold for secretin release of about 0.3 acid mmol/min suggests that secretin acts in a reserve capacity, stimulating the flow of pancreatic bicarbonate only when high levels of acidity require it.

8262 GASTROESOPHAGEAL SPHINCTER PRESSURE AND SERUM GASTRIN: REACTION TO FOOD STIMULATION IN NORMAL SUBJECTS AND IN PATIENTS WITH GASTRIC OR DUODENAL ULCER. (Eng.) Csendes, A.; Oster, M.; Brandsborg, O.; Moller, J.; Amdrup, E. (Hospital J. J. Aquirre, Santos Dumont 999, Santiago, Chile). *Scand. J. Gastroenterol.* 13(3):363-368; 1978.

The effect of solid food on gastroesophageal sphincter pressure (GESP) and serum gastrin concentration (SGC) was studied in patients with gastric ulcers (GU), duodenal ulcers (DU), and in normal controls. GESP and SGC measurements were made in the resting state and then followed for 2 hr after the ingestion of a normal mixed meal (29 g of protein, 28 g of fat, and 2 g of carbohydrates), a potato meal (3 g protein, 8 g fat, and 38 g carbohydrate), or an i.v. infusion of Aminosol (70% amino acids and 30% small peptides; total protein, 25 g). No significant differences were demonstrated in the basal GESP or SGC values among the three groups. After the intake of the protein meal, GESP rose 2.6 ± 1.2 mm Hg/min in controls and 5.0 ±

2.9 mm Hg/min in DU patients, while the SGC rose 19.5 ± 6.2 and 27 ± 7 pg/ml/min, resp. These nearly parallel increases are thought to be inter-related and due to the presence of intraluminal proteins in the upper gastrointestinal tract. The protein meal-stimulated GESP was significantly ($p<0.05$) higher than basal GESP from 60 to 100 min after eating (peak at 80 min in controls and at 60 min in DU patients). In GU patients, a decrease in GESP (-7.0 ± 1.4 mm Hg/min) and a rise in SGC (125 ± 41 pg/ml/min) occurred after protein meal ingestion. The decrease was significantly ($p<0.05$) lower than basal values from 60 to 100 min postprandially (peak decrease at 80 min). The increase in SGC was significant ($p<0.05$) in GU patients at 30 and 45 min, while in DU patients, it was significant ($p<0.05$) at 15, 30, and 45 min postprandially. There was no variation in SGC compared with basal values in any subject after ingestion of the potato meal or during the i.v. infusion of Aminosol. When the potato meal was given to two controls and two DU patients, the GESP decreased (peak decrease at 40 min, returning to basal values at 80 min). The i.v. infusion of Aminosol in a normal and a DU patient produced no GESP changes. The results show that the effect of protein food on GESP is different for GU and DU patients, possibly due to an alteration in the intrinsic properties of the specialized sphincteric muscle. The effect of food is mediated by proteins in the gastrointestinal tract.

8263 A COMPARATIVE STUDY OF DOUBLE CONTRAST AND SINGLE CONTRAST BARIUM MEALS WITH ENDOSCOPIC ARBITRATION IN THE DIAGNOSIS OF PEPTIC ULCER. (Eng.) Lavelle, M. I.; Venables, C. W.; Douglas, A. P.; Thompson, M. H.; Owen, J. P.; Hacking, P. M. (Royal Victoria Infirmary, Newcastle upon Tyne, England). *Clin. Radiol.* 28(6):625-627; 1977.

8264 LONG-TERM PROGNOSIS OF DUODENAL ULCER: FOLLOW-UP STUDY AND SURVEY OF DOCTORS' ESTIMATES. (Eng.) Greibe, J.; Bugge, P.; Gjorup, T.: Lauritzen, T.; Bonnevie, O.; Wulff, H. R. (Herlev Univ. Hosp., 2730 Herlev, Copenhagen, Denmark). *Br. Med. J.* 2(6102):1572-1574; 1977.

8265 SURGICAL TREATMENT OF CHRONIC DUODENAL ULCER DISEASE. (Eng.) O'Leary, J. P. (Coll. Medicine, Univ. Florida, Box J-286, Gainesville, FL 32610). *Am. Surg.* 44(2):108-112; 1978.

8266 RELATION OF THE ACID CONCENTRATION OF THE PARIETAL COMPONENT OF THE GASTRIC JUICE TO THE MAXIMAL ACID OUTPUT IN MAN. (Eng.) Ligny, G.; Smets, Ph. (Hopital Civil, Boulevard Janson, 6000 Charleroi, Belgium). *Biomedicine* 26(6):396-399; 1977.

8267 A FIBREENDOSCOPIC STUDY OF ACUTE UPPER GASTROINTESTINAL HEMORRHAGE IN NAIROBI, KENYA. (Eng.) Hansen, D. P.; Daly, D. S. (Dept. Medicine, Univ. California--San Diego, 225 W. Dickinson St., San Diego, CA 92103). *Am. J. Trop. Med. Hyg.* 27(1):197-200; 1978.

8268 SURGICAL TREATMENT OF THE BLEEDING PEPTIC ULCER. (Eng.) Kalsbeek, H. L. (Dept. Surgery, Diakonessen-inrichting Bronovo, The Hague, Netherlands). *Arch. Chir. Neerl.* 29(4):229-238; 1977.

8269 3. PRACTICAL MANAGEMENT OF RECURRENT PEPTIC ULCER. (Eng.) Cohen, M. M. (Vancouver General Hosp., Vancouver, British Columbia V5Z 1L5, Canada). *Can. J. Surg.* 21(1):21-24; 1978.

8270 2. VAGOTOMY AND ITS VARIATIONS. (Eng.) Doris, P. J. (Hotel-Dieu Hosp., Kingston, Ontario K7L 3H6, Canada). *Can. J. Surg.* 21(1):19-20; 1978.

8271 EFFECTS OF INTRAVENOUS TRITHIOZINE (I.S.F. 2001) ON GASTRIC SECRETION IN MAN. (Eng.) Corinaldesi, R.; Luchetta, L.; Ricci, P.; Guidoboni, R.; Casadio, R.; Fabbri, R.; Barbara, L. (Dept. Gastroenterology, Univ. Bologna, Bologna, Italy). *Panminerva Med.* 19(5):333-338; 1977.

8272 CROSS-OVER CLINICAL COMPARISON BETWEEN ANTI-SECRETORY ACTIVITY OF PROPANTHELINE BROMIDE AND TRITHIOZINE (I.S.F. 2001). (Eng.) Corinaldesi, R.; Luchetta, L.; Ricci, P.; Guidoboni, R.; Casadio, R.; Fabbri, R.; Barbara, L. (Dept. Gastroenterology, Univ. Bologna, Bologna, Italy). *Panminerva Med.* 19(5):339-344; 1977.

8273 SERUM GASTRIN LEVELS IN THE DIFFERENTIAL DIAGNOSIS OF RECURRENT PEPTIC ULCERATION DUE TO RETAINED GASTRIC ANTRUM. (Eng.) Webster, M. W.; Barnes, E. L.; Stremple, J. F. (Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA). *Am. J. Surg.* 135(2):248-252; 1978.

8274 EFFECT OF HIGHLY SELECTIVE VAGOTOMY ON GASTRIC MOTOR ACTIVITY OF DUODENAL ULCER PATIENTS. (Eng.) Bortolotti, M.; Labo, G.; Serantoni, C.; Ciani, P. (Dept. Clinical Medicine, Univ. Bologna, Bologna, Italy). *Digestion* 17(2):108-120; 1978.

8275 GASTRIC EMPTYING AFTER DIFFERENT SURGICAL PROCEDURES FOR DUODENAL ULCER. (Eng.) Faxen, A.; Berger, T.; Kewenter, J.; Kock, N. G. (Sahlgren's Hosp., Univ. Goteborg, Goteborg, Sweden) *Scand. J. Gastroenterol.* 12(8):983-987; 1977.

8276 GASTRIC RESPONSE TO MEAT EXTRACT STIMULATION IN PATIENTS WITH GASTRODUODENAL ULCER AND PATIENTS AFTER VAGOTOMY OR ANTRECTOMY. (Eng.) Tani, M.; Shimazu, H.; Takahashi, T.; Asakuma, S.

(Univ. Tokyo Faculty Medicine, Tokyo, Japan). *Jpn. J. Surg.* 8(1):10-18; 1978.

8277 ENDOSCOPIC AND RADIOLOGICAL ASSESSMENT OF RECURRENT ULCERATION AFTER PEPTIC ULCER SURGERY. (Eng.) Salter, R. H.; Girdwood, T. G.; Scott-Harden, W. G.; Cole, T. P. (Cumberland Infirmary, Carlisle, England). *Br. J. Radiol.* 51(604):257-259; 1978.

8278 RESULTS OF ELECTIVE DUODENAL ULCER SURGERY IN WOMEN: COMPARISON OF TRUNCAL VAGOTOMY AND ANTRECTOMY, GASTRIC SELECTIVE VAGOTOMY AND PYLOROPLASTY, PROXIMAL GASTRIC VAGOTOMY. (Eng.) Herrington, J. L., Jr.; Sawyers, J. L. (Dept. Surgery, Vanderbilt Univ., Nashville, TN). *Ann. Surg.* 187(5):576-582; 1978.

8279 PLACEBOS, ANTACIDS AND CIMETIDINE FOR DUODENAL ULCER. (Eng.) Fordtran, J. S. (Univ. Texas Southwestern Medical Sch., Dallas, TX 75235). *N. Engl. J. Med.* 298(19):1081-1083; 1978.

8280 THE AARHUS COUNTY VAGOTOMY TRIAL. I. AN INTERIM REPORT ON PRIMARY RESULTS AND INCIDENCE OF SEQUELAE FOLLOWING PARIETAL CELL VAGOTOMY AND SELECTIVE GASTRIC VAGOTOMY IN 748 PATIENTS. (Eng.) Amdrup, E.; Andersen, D.; Hostrup, H. (Kommunehospitalet, 8000 Aarhus C., Denmark). *World J. Surg.* 2(1):85-90; 1978.

8281 THE AARHUS COUNTY VAGOTOMY TRIAL. II. AN INTERIM REPORT ON REDUCTION IN ACID SECRETION AND ULCER RECURRENCE RATE FOLLOWING PARIETAL CELL VAGOTOMY AND SELECTIVE GASTRIC VAGOTOMY. (Eng.) Andersen, D.; Hostrup, H.; Amdrup, E. (Kommunehospitalet, 8000 Aarhus C., Denmark). *World J. Surg.* 2(1):91-100; 1978.

8282 RUPTURE OF THE SPLEEN IN A PATIENT WITH A PERFORATED DUODENAL ULCER AND INFECTIOUS MONONUCLEOSIS. (Eng.) Gray, R. (King's Coll. Hosp., London SE5, England). *Postgrad. Med. J.* 54(627): 51-55; 1978.

See also, 7974, 8045, 8128, 8165, 8167, 8171, 8175, 8181, 8206, 8224, 8230, 8239, 8249, 8251, 8255, 8363, 8382, 8402, 8572, 8596.

SMALL INTESTINE

8283 PARAPROTEINEMIA IN NORMAL FAMILY MEMBERS OF EIGHT CASES WITH PRIMARY INTESTINAL LYMPHOMAS IN IRAQ. (Eng.) Alsabti, E. A. K. (Albaath Specific Protein Reference Unit, Baghdad, Iraq). *Oncology* 35(2):68-72; 1978.

The clinical and laboratory findings in eight patients with alpha-chain disease associated with primary intestinal lymphomas were analyzed, and the serum IgA level was determined in the families of these patients. The clinical, laboratory, radiological, and histopathological findings in the eight patients suggested malabsorption. Abdominal pain, diarrhea, and weight loss were present in all patients. The villous architecture was distorted in all patients; sparse crypts, pleomorphism and hypercellularity of the lamina propria, invasion of the muscularis mucosa, and lymphatic dilatation were present in six. The serum IgA levels ranged between 380 and 1,405 mg/100 ml. The serum IgG level was abnormally low (170-420 mg/100 ml) in all but one patient (1,120 mg/100 ml). The IgM levels ranged between 0 and 425 mg/100 ml. IgD levels were normal in all cases, ranging from 0 to 9.2 mg/100 ml. Of the 129 family members, 23 had abnormal IgA levels. This result suggests that these individuals should be carefully monitored for the development of intestinal lymphoma.

8284 CROHN'S DISEASE: "ITS GYNECOLOGIC ASPECT." (Eng.) Donaldson, L. B. (Medical Dental Bldg., Seattle, WA 98101). *Am. J. Obstet. Gynecol.* 131(2):196-202; 1978.

The medical records of 103 patients with gynecologic features of Crohn's disease are reviewed. The clinical history most frequently included signs and symptoms related to bowel obstruction. Specific signs and symptoms included abdominal pain in 63 cases; abscesses in 36 (13 pelvic, 11 perirectal, 8 perineal, 2 ischiorectal, 1 gluteal fold, and 1 epidural); tender adnexal mass in 28; rectal or perineal pain in 18; rectal bleeding in 12; vulvovaginitis, rectal fissure, or perineal ulcer in 8 cases each; right abdominal mass in 6; and dyspareunia in 4. The earliest signs and symptoms in some instances appeared to be unrelated to Crohn's disease and consisted of infections, ulcers, fissures, abscesses, and fistulas of the internal and external pelvic structures. Of 55 patients misdiagnosed, a large number had these prodromal gynecologic aspects of the disease. In over half of the 103 patients with Crohn's disease, intestinal obstruction was considered in the initial diagnosis. The diagnosis of the disease was first established at laparotomy in 23 of the 103 patients. The incorrect preoperative diagnoses in most in-

stances were appendicitis (27), pelvic inflammatory disease (11), or ovarian cysts (5). There were 27 appendectomies performed, but there were no cases of acute appendicitis. The present study indicates no unfavorable effect of Crohn's disease on pregnancy; the effect of pregnancy on Crohn's disease appears to be minimal, with exacerbation of the disease occurring in only two patients in the immediate postpartum period. It is suggested that the gynecologic aspect of Crohn's disease should be considered in making a diagnosis, especially when abdominal pains and bowel complaints are present.

8285 JEJUNO-GASTRIC INTUSSUSCEPTION--GASTROSCOPIC DIAGNOSIS. (Eng.) Varley, C. C.; Dyer, N. H. ("Stanhill," 34 Queens Rd., Melbourne, Victoria 3004, Australia). *Aust. N.Z. J. Med.* 7(5): 515-517; 1977.

The case of a patient in whom jejuno-gastric intussusception was diagnosed by gastroscopy is presented. The patient was a 76-yr-old woman who had undergone a Polya gastrectomy for pyloric stenosis complicating a duodenal ulcer 8 yr previously. She presented after developing colicky abdominal pain, projectile vomiting, and hematemesis. There was mild epigastric tenderness on examination. Gastroscopy revealed normal inflamed gastric mucosa of a Polya gastrectomy. At the stoma, the lumen of the afferent limb was patent and contained bile-stained fluid. The efferent limb lumen was filled with intussuscepted small bowel, which had a grossly swollen, engorged bleeding surface. The intussuscepted bowel extended nearly up to the hiatus. At laparotomy, the intussusception was reduced manually, and the patient had an uncomplicated recovery. Endoscopy is the method of choice to diagnose this uncommon, but important condition.

8286 ADAPTATIVE CHANGES IN THE SMALL INTESTINE FOLLOWING BYPASS OPERATION FOR OBESITY: A RADIOLOGICAL AND HISTOLOGICAL STUDY. (Eng.) Solhaug, J. H.; Tvete, S. (Sjukhuset, 68500 Torsby, Sweden). *Scand. J. Gastroenterol.* 13(4):401-408; 1978.

Roentgenographic follow-up studies were made in seven patients before and at various intervals after jejunoileal bypass, to investigate adaptive changes to this procedure. Length measurements were made by a modified intubation technique, and intestinal transit time was estimated after a standard contrast meal. At surgery, 20 cm of proximal jejunum was anastomosed end-to-end to the distal 25 cm of ileum. With increasing observation time, a successive increase was observed in intestinal length both in the jejunal and ileal segments. One year after bypass, the mean growth of the functioning ileum was 100%, and the elongation of the jejunal segment was 50%. Eighteen months after the bypass, the mean growth values for the ileum and jejunum were 128% and 80%, resp. In serial films taken 4-6, 12, and 18 months after bypass, the diameter of both the functioning jejunum and ileum remained essentially unaltered. The mean increase in the diameter of the jejunum and ileum before and after bypass was approximately 40% and 50%, resp. The mean transit time of a test meal from the pylorus to the anastomotic level at 6, 12, and 18 months was 1.6 ± 0.6, 4.6 ± 6, and 4.8 ± 5 min, resp. The transit time from the anastomosis to the cecum was 38 ± 29, 55 ± 26, and 60 ± 28 min, resp. The postoperative gastric and functioning small intestinal emptying times were generally unaltered compared with preoperative findings. The mean increase in villous height in the functioning jejunum was 14% at 6 months after bypass and 55% after 12 months. In the ileum, the values were 32% and 66%, resp. In the bypassed ileum, the mean villous height decreased by 16% at 6 and 12 months. The observations suggest that there is an increase in intestinal absorptive surface following small intestinal bypass, which is due to dilatation and elongation of the functioning remnant as well as to an increase in villous height. An increase in transit time, permitting prolonged contact between nutrients and the absorptive intestinal surface, is probably a contributing factor in the leveling off of weight loss observed between 12 and 18 months after bypass.

8287 PEUTZ-JEGHERS SYNDROME: EXPERIENCE WITH TWENTY PATIENTS IN FIVE GENERATIONS. (Eng.) McAllister, A. J.; Richards, K. F. (511 Medical Arts Bldg., Salt Lake City, UT 84111). *Am. J. Surg.* 134:717-720; 1977.

Experience with 20 patients in one family with the Peutz-Jegher's syndrome is reviewed, and the case report of a patient with an unusual presentation is given. The disease is characterized by pigmented spots on the lips and oral mucosa and intestinal polyposis. The intestinal polyps are hamartomas, and occur throughout the gastrointestinal system, with the heaviest incidence in the jejunum. In the family reported, which represents five generations, only three members were without symptoms. The clinical course of this syndrome is characterized by asymptomatic periods interspersed with complications such as intermittent cramping, abdominal pain, small intestinal intussusception, and hemorrhage. The patient whose case report is presented was unusual in that colonic intussusception was present. Examination revealed no abdominal masses, and bowel sounds were absent. Radiologic studies established the diagnosis of colonic intussusception. At surgery, 10 cm of the transverse colon was intussuscepted with a large leading polyp, measuring 5.5 x 4.5 x 4.0 cm, which was removed together with 11 cm of involved bowel. These observations suggest that intussusception can occur at any level of the gastrointestinal tract. The hematocrit should be checked every 6 months in patients with the Peutz-Jegher's syndrome to test for occult bleeding. X-ray films should be taken every 2 yr after the age of 10, and any polyps visualized in the stomach or colon should be removed endoscopically.

8288 RIGHT-SIDED DIAPHRAGMATIC HERNIA: NEONATAL PRESENTATION ASSOCIATED WITH AN UNUSUAL PHYSICAL SIGN. (Eng.) Edelsten, A. D.; Tuck, S. (Royal Devon and Exeter Hosp., Exeter, England). *Br. Med. J.* 2(6099):1392-1393; 1977.

8289 SMALL-INTESTINAL OBSTRUCTION FROM A RICHTER'S HERNIA AT THE SITE OF INSERTION OF A LAPAROSCOPE. (Eng.) Bourke, J. B. (General Hosp., Nottingham, England). *Br. Med. J.* 2(6099):1393-1394; 1977.

8290 PNEUMATOSIS INTESTINALIS. (Eng.) Meyers, M. A.; Ghahremani, G. G.; Clements, J. L., Jr.; Goodman, K. (New York Hosp., 525 E. 68th St., New York, NY 10021). *Gastrointest. Radiol.* 2(2): 91-105; 1977.

8291 ISCHEMIC JEJUNITIS AND PNEUMATOSIS INTESTINALIS SECONDARY TO ANASTOMOTIC OBSTRUCTION FOLLOWING JEJUNAL ATRESIA REPAIR. (Eng.) Kleinman, P. L.; King, D. R. (Natl. Naval Medical Center, Bethesda, MD 20014). *Gastrointest. Radiol.* 2(2):113-115; 1977.

8292 POLYPOID GASTRIC HETEROTOPIA CAUSING SMALL BOWEL INTUSSUSCEPTION. (Eng.) Santos, R.; Wills, J. S. (Wilmington Medical Center, Box 1668, Wilmington, DE 19899). *Gastrointest. Radiol.* 2(2):123-124; 1977.

8293 KNOTTING AND PERFORATION OF A MECKEL'S DIVERTICULUM. (Eng.) Desai, S.; Cook, R. C. M. (Preston Royal Infirmary, Deepdale Rd., Preston, Lancashire, England). *Z. Kinderchir.* 22(2):175-180; 1977.

8294 SPONTANEOUS RESOLUTION OF A MESENTERIC FIBROMATOSIS. (Eng.) Sturzaker, H. G.; Berry, C. L.; McColl, I. (Guy's Hosp., London, England). *J. R. Coll. Surg. Edinb.* 22(6):395-399; 1977.

8295 DUODENAL HODGKIN'S DISEASE. (Eng.) Balthazar, E. J. (Metropolitan Hosp. Center, 1901 First Ave., New York, NY 10029). *Am. J. Gastroenterol.* 68(3):306-311; 1977.

8296 PARADUODENAL HERNIAS. (Eng.) Bell-Thomson, J.; Vieta, J. O.; Yiavasis, A. A. (Lenox Hill Hosp., 100 E. 77th St., New York, NY 10021). *Am. J. Gastroenterol.* 68(3):254-259; 1977.

8297 DUODENAL VARICES: A RARE CAUSE OF UPPER GASTROINTESTINAL HEMORRHAGE. (Eng.) Schoettle, G. P., Jr.; Davis, W. D., Jr.; Bowen, J. C. (Ochsner Clinic, 1514 Jefferson Highway, New Orleans, LA 70121). *J. La. State Med. Soc.* 130(1):7-10; 1978.

8298 ENTERITIS CYSTICA PROFUNDA. (Eng.) Kyriakos, M.; Condon, S. C. (Barnes Hosp., Barnes Hosp. Plaza, St. Louis, MO 63110). *Am. J. Clin. Pathol.* 69(1):77-85; 1978.

8299 METABOLIC CHANGES AFTER JEJUNO-ILEAL BYPASS FOR OBESITY. (Eng.) Solhaug, J. H.; Grundt, I. (Dept. Surgery, Univ. Bergen, Bergen, Norway). *Scand. J. Gastroenterol.* 13(2): 169-175; 1978.

8300 PREDUODENAL PORTAL VEIN: TWO CASES WITH DIFFERING PRESENTATION. (Eng.) Stevens, J. C.; Morton, D.; McElwee, R.; Hamit, H. F. (Charlotte Memorial Hosp., Charlotte, NC 28234). *Arch. Surg.* 113(3):311-313; 1978.

8301 RISKS OF SMALL BOWEL BIOPSY TECHNIQUES [Abstract]. (Eng.) Tytgat, G. (No affiliation given). *Scand. J. Gastroenterol.* 12 (Suppl. 47):18-19; 1977.

8302 A POSSIBLE CASE OF HYPOTHALAMIC HYPOFUNCTION CAUSED BY PROLONGED MALNUTRITION DUE TO INTESTINAL BLIND LOOP. (Eng.) Nakashima, T.; Fuchigami, T.; Inoue, K.; Omae, T. (Faculty Medicine, Kyushu Univ., Fukuoka 812, Japan). *Endocrinol. Jpn.* 24(6):529-536; 1977.

8303 EFFECT OF PREPARATION OF THE SMALL INTESTINE ON MICROFLORA AND POSTOPERATIVE WOUND INFECTION. (Eng.) Parker, T. H.; O'Leary, J. P. (Shands Teaching Hosp., Univ. Florida, Gainesville, FL). *Surg. Gynecol. Obstet.* 146(3):379-382; 1978.

See also, 8040, 8136, 8141, 8142, 8158, 8163, 8168, 8169, 8170, 8171, 8175, 8188, 8200, 8203, 8210, 8239, 8248, 8251, 8304, 8305, 8307, 8314, 8318, 8359, 8361, 8374, 8398, 8507, 8508, 8564, 8571, 8573, 8574, 8575, 8577, 8601, 8602, 8606, 8619.

8304 STANDARDIZED ('TRIFIXED') DIET IN THE STUDY OF CHRONIC MALABSORPTION SYNDROMES. (Eng.) Hylander, E.; Jarnum, S.; Keldsbo, I. L.; Thale, M. (Rigshospitalet, 2100 Copenhagen O, Denmark). *Scand. J. Gastroenterol.* 13(4):423-431; 1978.

Intestinal absorption was studied in patients with different types of malabsorption syndromes who were placed on diets with fixed amounts of calcium, oxalate, and fat. The patients included 70 with Crohn's disease, 11 with ulcerative colitis, 40 who had undergone an intestinal bypass operation, 9 with nontropical sprue, 10 with short-bowel syndrome, and 3 with other gastrointestinal diseases. The patients received 70 g of fat, 800 mg of calcium, and 200 mg of oxalate daily in their diet. Fecal fat, ^{47}Ca absorption, ^{14}C-oxalate absorption, and renal oxalate excretion were measured, and in the majority of patients a ^{14}C-glycocholic acid breath test was also performed. In 83 patients in whom calcium absorption was determined by whole-body counting and by the accumulation of absorbed ^{47}Ca in the skeleton of the underarm, ^{47}Ca absorption was almost identical ($r=0.92$, $p<0.001$) by the two procedures. ^{14}C-oxalate absorption and renal oxalate excretion in the 138 patients in whom both procedures were carried out also correlated well ($r=0.85$, $p<0.001$). Fecal fat and renal oxalate excretion were weakly correlated ($r=0.63$, $p<0.001$), but there was no correlation between fecal fat and calcium absorption or between oxalate and calcium absorption. It is recommended that the "trifixed" regimen with absorption studies of fat, calcium, and oxalate be undertaken before treatment for the reduction of steatorrhea or hyperoxaluria or for improvement of calcium absorption in chronic malabsorption syndromes.

8305 SMALL-BOWEL MALABSORPTION AND GASTROINTESTINAL MALIGNANCY. (Eng.) Collins, S. M.; Hamilton, J. D.; Lewis, T. D.; Laufer, I. (McMaster Univ. Medical Center, Hamilton, Ontario, Canada). *Radiology* 126(3):603-609; 1978.

The cases of four patients with malabsorption and gastrointestinal malignancies are presented. All patients demonstrated histological and radiological features of celiac disease and showed improvement on gluten withdrawal. The first patient had been asymptomatic for 27 yr after experiencing symptoms of malabsorption, diagnosed as tropical sprue. He developed diarrhea, steatorrhea, anorexia, lethargy, and weight loss, which resolved upon withdrawal of dietary gluten. A squamous cell carcinoma of the middle third of the esophagus was discovered 2 yr later; no abnormality could be detected on the esophagrams taken 2 yr earlier. The second patient had a childhood history of celiac disease but had been relatively asymptomatic on a normal diet since the age of 15 yr. At the age of 47, he developed severe steatorrhea, weight loss, finger clubbing, and abdominal distention, and the symptoms did not improve on gluten withdrawal, even when prednisone was added to the regimen. A high serum IgA level was present, suggesting a lymphoma. Small bowel biopsy returned to normal and marked clinical improvement occurred when vincristine and cyclophosphamide were added to the regimen. A small bowel enema study suggested extrinsic ileal compression, and an unresectable, mesenteric, poorly differentiated reticulum cell sarcoma was discovered. The third patient presented with a 1-yr history of symptoms of malabsorption at age 74. Subtotal villous atrophy persisted, despite a 3-month period of gluten withdrawal. Following the removal of an *in situ* jejunal adenocarcinoma, there was marked clinical improvement while the patient was on a gluten-free diet, and a jejunal biopsy showed considerable improvement. The last patient, a 55-yr-old man, developed celiac disease that was initially responsive to gluten withdrawal; he became unresponsive with the development of pancreatic carcinoma. With a combination of prednisone and gluten withdrawal, he was once again rendered gluten-sensitive, as demonstrated by his marked clinical improvement and by the dramatic deterioration that occurred when gluten was accidentally reintroduced. It appears that there is an increased incidence of gastrointestinal malignancy in patients with celiac disease. These patients should have regular radiographic surveys.

8306 MALABSORPTION IN INFECTIOUS MONONUCLEOSIS. (Eng.) LoIudice, T. A.; Lang, J. A. (Cedar-Locust Medical Pavilion, Ste. 206, 157 W. Cedar St., Akron, OH 44307). *Am. J. Gastroenterol.* 67(6):604-607; 1977.

The case of a patient in whom infectious mononucleosis was complicated by malabsorption is presented. The patient, a 23-yr-old man, presented with a fever of 40 C, cramping abdominal pain, nausea, vomiting, diarrhea, polyarthralgias, light-headedness, irritability, malaise, anorexia, and weight loss. A mono spot test was positive and a heterophil titer was 1:1,448. When diarrhea continued for longer than 1 week, further investigations were undertaken. The results of these included a d-xylose excretion (25 g p.o. dose) of 2.5 g/5 hr, a 72-hr stool fat of 36 g, and a duodenal aspirate that revealed no organisms. In addition, a small bowel roentgenographic series showed extensive thickening of mucosal folds with puddling of barium, and a jejunal biopsy revealed marked blunting of villi with an inflammatory infiltrate in the lamina propria. The patient responded to treatment consisting of i.v. fluid and electrolyte replacement, i.m. codeine, and bed rest. Two months after admission, a mono spot test was negative and the results of a small bowel series and a repeat small bowel biopsy had returned to normal. Malabsorption appears to be a rare complication of this disease.

8307 COMPLETE CLINICAL, HISTOLOGICAL, AND IMMUNOLOGICAL REMISSION IN A PATIENT WITH ALPHA-CHAIN DISEASE TREATED BY ORAL ANTIBIOTICS. (Fre.) Rambaud, J. C.; Piel, J. L.; Galian, A.; Leclerc, J. P.; Danon, F.; Girard-Pipeau, F.; *et al.* (Hopital Saint-Lazare, 107 bis, rue du Faubourg-Saint-Denis, F 75475 Paris Cedex 10, France). *Gastroenterol. Clin. Biol.* 2(1):49-61; 1978.

A case is presented of alpha-chain disease in a 17-yr-old Algerian boy who had no history of immunological disease in his family (parents and five siblings). Initial symptoms were fever, vomiting, diarrhea, and abdominal pain. All symptoms except diarrhea subsided at first, but latter grew progressively worse with weight loss, edema of lower limbs, and finally tetany. Rectal and gastric biopsies obtained by endoscopy suggested the diagnosis of alpha-chain disease (plasma cell infiltrations of mucosal crypts) that was confirmed by immunological studies. The patient was treated by various antibiotics (tetracycline, metronidazole, ampicillin, colistine, and amphotericine) over a 3-month period. After only 12 days on antibiotics, intestinal malabsorption improved and the abnormal IgA disappeared from the serum but not from the intestinal cellular infiltrate. Restoration of the severe magnesium depletion resulted in normal blood calcium levels and disappearance of tetany. A *Giardia lamblia* infection was cured at the same time. The complete remission of symptoms was confirmed by biopsy performed at laparotomy 6 months after end of antibiotic treatment. Proximal jejunal biopsy showed normal plasma cells with a moderate increase of the alpha-chain IgA cells compared with the normal state.

8308 SHORT-TERM GLUTEN CHALLENGE TEST IN CELIAC DISEASE. (Ita.) Mastella, G.; Trabucchi, C.; Mengoli, V.; Olivieri, D.; Rossi, F. A. (Ospedale Infantile Alessandri, Verona, Italy). *Minerva Pediatr.* 29(37):2235-2252; 1977.

An analysis of the short-term gluten challenge test in celiac disease is presented. Ninety-one children (aged 12 months-10 yr) were divided into three groups: 29 diagnosed as having celiac disease (group 1), 45 patients with chronic diarrhea of unknown etiology (group 2), and 17 with recurrent intestinal disturbances (group 3). All received gluten for 9-17 days either as commercial gluten (800 mg/kg intradermally/day) or in normal alimental form (10-25 g/day). The body weight, number and character of defecations, circumference of the abdomen, mood and physical activity, general clinical results, biopsy of the intestinal mucosa, xylose absorption test, and levels of IgA, IgG, IgM, and the C'3 complement fraction were monitored over a 20-day period. The condition of the intestinal mucosa was numerically evaluated over the range from 0-3 (grade 3 lesion) to 10-12 (normal mucosa). The average values before treatment were 5.29 (group 1), 7.55 (group 2), 9.24 (group 3), and after treatment were 3.48 (group 1, $p<0.05$), 6.87 (group 2), and 9.05 (group 3). The xylose absorption test showed positive but insignificant differences after treatment in 20/32 tests in group 1, 21/45 tests in group 2, and 4/19 tests in group 3. Changes in the coefficient of absorption of fat (C.A.%) before and after treatment were 91.92 to 90.81 C.A.% in group 1, 93.91 to 94.75 C.A.% in group 2, and 96.58 to 97.13 C.A.% in group 3; positive changes occurred in 17/33 tests in group 1, 10/52 tests in group 2, and 1/19 tests in group 3. The daily weight of the feces increased from 77.03 g to 122.76 g, $p<0.01$ in group 1, from 88.37 to 89.73 in group 2, and from 97.71 to 99.26 in group 3. Differences between groups 1 and 3 were significant for all four tests ($p<0.01$); between groups 1 and 2 were significant for the condition of the intestinal mucosa ($p<0.001$), C.A.% of fat ($p<0.01$), and weight of feces ($p<0.05$); and between groups 2 and 3 were significant only for the xylose test ($p<0.05$). On the basis of the gluten test, the diagnosis was celiac disease in group 1 patients, 6/45 group 2 patients, and 0/17 group 3 patients; no diagnosis could be reached on this basis in 4/29, 15/45, and 1/17 in groups 1, 2, and 3, resp. The variability of results with time in some patients suggests that the response to gluten is not constant, thus leaving a margin of uncertainty in a number of patients suspected of celiac disease. The results of the measurements of the immunoglobulins and C'3 fraction suggest that gluten does not have an immunogenic effect in these patients. It was concluded that the short-term gluten test is well tolerated in these patients and remains a useful test until a more definite one is found.

8309 TOLERANCE TO LACTOSE AMONG LACTASE-DEFICIENT AMERICAN INDIANS. (Eng.) Newcomer, A. D.; McGill, D. B.; Thomas, P. J.; Hofmann, A. F. (Mayo Clinic and Mayo Foundation, Rochester, MN 55901). *Gastroenterology* 74(1): 44-46; 1978.

8310 GIANT AXONAL NEUROPATHY: POSSIBLY SECONDARY TO VITAMIN B_{12} MALABSORPTION. (Eng.) Schochet, S. S., Jr.; Chesson, A. L., Jr. (Univ. Texas Medical Branch, Galveston, TX 77550). *Acta Neuropathol. (Berl.)* 40(1):79-83; 1977.

8311 DUAL OCCURRENCE OF CELIAC SPRUE AND IDIOPATHIC PORTAL HYPERTENSION. (Eng.) Drinka, P. J.; Imbeau, S. A. (Veterans Admin. Hosp., Madison, WI). *Wis. Med. J.* 76(11):S134-S136; 1977.

8312 CLINICAL MEASUREMENT OF IRON ABSORPTION. (Eng.) Cook, J. D.; Lipschitz, D. A. (Kansas Univ. Sch. Medicine, Kansas City, KS). *Clin. Hematol.* 6(3):567-581; 1977.

8313 CHRONIC CALCIFIC PANCREATITIS IN ASSOCIATION WITH CELIAC DISEASE. (Eng.) Pitchumoni, C. S.; Thomas, E.; Balthazar, E.; Sherling, E. (Metropolitan Hosp. Center, 1901 First Ave., New York, NY 10029). *Am. J. Gastroenterol.* 68(4):358-361; 1977.

8314 CALCIUM MALABSORPTION AND ABSENCE OF BONE DECALCINATION FOLLOWING INTESTINAL SHUNT OPERATION FOR OBESITY: A COMPARISON OF TWO TYPES OF OPERATION. (Eng.) Dano, P.; Christiansen, C. (Rigshospitalet, Copenhagen, Denmark). *Scand. J. Gastroenterol.* 13(1):81-85; 1978.

8315 POSSIBILITIES OF DIFFERENTIATION BETWEEN MORBID AND INVOLUTIVE CHANGES IN ENTERAL RESORPTION. COMMUNICATION 1: MORBID CHANGES OF ENTERAL RESORPTION. (Ger.) Weiner, R.; Laue, R.; Dietze, F.; Schone, D.; Weiner, C. (Bezirkskrankenhauses St. Georg, Str. der DSF 141, DDR-7021 Leipzig, E. Germany). *Dtsch. Gesundheitsw.* 32(30):1423-1428; 1977.

8316 MALABSORPTION SYNDROME IN CHRONIC LYMPHOCYTIC LEUKEMIA: ENDOSCOPY OF THE UPPER SEGMENT OF THE SMALL INTESTINE. (Ger.) Cottier, C.; Fankhauser, S.; Clemencon, G. (Kantonsspital Olten, Olten, Switzerland). *Z. Gastroenterol.* 15(10):641-642; 1977.

See also, 8161, 8163, 8168, 8170, 8302, 8367, 8403, 8406, 8565, 8567, 8610.

LARGE INTESTINE

8317 SYSTEMIC PROPHYLAXIS WITH DOXYCYCLINE IN SURGERY OF THE COLON AND RECTUM. (Eng.) Hojer, H.; Wetterfors, J. (Regionsjukhuset, S-581 85 Linkoping, Sweden). *Ann. Surg.* 187(4):362-368; 1978.

A prospective double-blind study on the effects of doxycycline as a prophylactic antimicrobial in elective colonic surgery was carried out in 118 patients. Fifty-eight patients received 200 mg of doxycycline, p.o., and 60 received placebo, 4-6 hr before surgery; 100 mg of doxycycline or placebo was given daily for 5 days postoperatively. A significantly lower incidence of abdominal wound sepsis was seen in the patients treated with doxycycline than in controls (8.6 versus 41.5%, $p<0.001$). Abdominal septic complications of grade 3 and 4 in severity were less frequent in the treatment group ($p<0.01$). Macroscopic peritoneal contamination occurred with the same incidence in both groups but the rate of associated wound or intra-abdominal sepsis dropped from 79% in the controls to 33% in the treated group. When the wound culture was negative upon closure, doxycycline treatment caused a significant ($p<0.002$) decrease in the rate of abdominal wound sepsis. Doxycycline treatment significantly reduced the rate of abdominal sepsis in malignant disease to 17% from 70% in the controls ($p<0.001$), and significantly reduced the rate of abdominal sepsis in both strictly intra- and mainly extraperitoneal procedures ($p<0.01$ in both cases). Perineal infection occurred in 20% of patients in the doxycycline-treated patients and in 47% of the controls; in the former group, it was the sole septic complication, whereas in the latter group, the infection was combined with some type of abdominal sepsis. Doxycycline treatment also reduced the rates of urinary tract infections and pulmonary complications ($p<0.02$ and $p<0.04$, resp.). None of the doxycycline-treated patients required repeat laparotomy due to septic complications, whereas eight patients in the control group required further surgery for sepsis. The results indicate that doxycycline treatment significantly reduces both the incidence and severity of postoperative sepsis in elective colorectal surgery.

8318 RISKS OF SURGERY OF THE GASTROINTESTINAL TRACT AFTER RADIATION OF THE ABDOMINAL AND PELVIC CAVITIES. (Fre.) Mendes da Costa, P.; Gerard, A. (Institut Jules-Bordet, 1, rue Heger-Bordet, 1000 Brussels, Belgium). *Ann. Chir.* 31(10): 866-870; 1977.

Two distinct groups were observed for complications after surgery preceded by radiation of the abdominal or pelvic cavities. Group I was composed of 47 patients who underwent amputation of the rectum by the abdomino-perineal route for adenocarcinomas. This group had been treated at random with 3,450 rads of radiation over a 19-day period with surgery following within 8 days of the last radiation treatment. Alternative random treatment was chemotherapy with 5-fluorouracil (1,500 mg/m^2) 4 days prior to surgery. Group II was composed of 42 patients all of whom underwent surgery of the gastrointestinal tract including colostomy of the rectum (14), resection and anastomoses (4), or liberation of adhesions of the small intestine (2), and resection and anastomoses or colostomy of the colon (8). These patients had been treated with 4,500-6,000 rads previously with an average delay between radiotherapy and surgery of 13.3 months. In 28/42 patients in group II, surgery was necessitated by a radiation lesion of the small intestine, colon, or rectum; in 13 patients, surgical intervention was performed because of residual or recurrent neoplasm. There were five postoperative deaths in group I and seven postoperative deaths in group II. Complications in group I included cystitis (14), infection of the perineum (11), abdominal abscess (5), necrosis of the colostomy (3), obstruction of the intestine (4), and a small intestine fistula (1). Complications in group II included infection (19), ab-

scess (7), failure of anastomoses (7), intestinal obstruction (5), fistula of the digestive tract (4), paralytic ileus (2), and necrosis of the colostomy (2). It is concluded that utero-ileal anastomoses are contraindicated in previously irradiated patients, but intestinal anastomoses may be performed if sufficient collateral circulation is present.

8319 NEONATAL SMALL LEFT COLON SYNDROME: ITS RELATIONSHIP TO AGANGLIONOSIS AND MECONIUM PLUG SYNDROME. (Eng.) Berdon, W. E.; Slovis, T. L.; Campbell, J. B.; Baker, D. H.; Haller, J. O. (Babies Hosp., 622 W. 168th St., New York, NY 10032). *Radiology* 125(2):457-462; 1977.

To clarify whether meconium plug syndrome, colon inertia, and neonatal small left colon syndrome can be distinguished from aganglionosis of the splenic flexure on radiographic and clinical grounds without recourse to rectal biopsy, experience with colon inertia and neonatal small left colon in 11 infants was reviewed and compared with other published reports and cases obtained by a hospital survey. Contrast enema studies of patients with colon inertia and those with small left colon syndrome were identical, showing distended loops of small bowel, right colon, and transverse colon with a sharp transition at the splenic flexure to a left-sided microcolon with a diameter of ≤1 cm; the rectum could be distended to a caliber of 2-3 cm while administering the enema. These radiographs were also identical to most of those published in cases of meconium plug syndrome; moreover, in many cases, meconium had been passed prior to the enema or else no plug was demonstrated. While the clinical presentation of these foregoing probably identical syndromes may be quite similar to that of aganglionosis of the splenic flexure (abdominal distention, full-term status, difficulty in passing meconium) and while all may have plain radiographic evidence of distended loops with frequent air/fluid levels and contrast enemas showing a transition zone, aganglionosis extending to the splenic flexure is readily differentiated from functional left-sided microcolon by the fact that aganglionic bowel is rarely reduced in caliber compared with normal bowel, whereas the microcolon has a caliber <1 cm. Whether or not to obtain a rectal biopsy in such cases must be based on the clinical response to the initial cleansing enemas and the subsequent course.

8320 SIGNIFICANCE OF CYTOLOGICAL INVESTIGATIONS IN THE DETECTION OF NEOPLASTIC CHANGES OF THE RECTOSIGMOID. (Ger.) Miczban, I.; Feher, M. (Poliklinika, Janos Korhaz, Budapest, Hungary). *Tijdschr. Gastroenterol.* 20(4):237-250; 1977.

Six hundred eleven consecutive exfoliative cytological investigations of the rectosigmoid were performed in 578 patients. The patients were also examined histologically and clinically. The cytology was negative in 536 cases, which all were found to be normal histologically. The cytology was doubtful in 13 cases; ulcerative colitis was diagnosed histologically in 11 of these cases and tumor in 2. Cytology showed polyp in 18 cases; the histological examinations of these patients revealed ulcerative colitis in 1 and polyp in 17. The cytology showed malignant changes in 11 cases; one of them was diagnosed histologically as ulcerative colitis, another as polyp, and 9 as tumor. The cytology showed definitely malignant changes in 9/11 histologically confirmed cases of tumor, but it was doubtful in the other 2 cases. All tumors were operable. The findings indicate the great diagnostic value of exfoliative cytology of the rectosigmoid, especially in combination with fiber rectoscopy.

8321 MULTIPLE CARCINOMAS OF THE LARGE BOWEL: A NATURAL EXPERIMENT IN ETIOLOGY AND PATHOGENESIS. (Eng.) Enker, W. E.; Dragacevic, S. (Pritzker Sch. Medicine, Chicago, IL). *Ann. Surg.* 187(1):8-11; 1978.

Theories concerning bile acids in colon carcinogenesis were tested by assuming that if bile acids were involved in colon cancer, the incidence of left-sided second primary cancers in patients with metachronous large bowel cancer would be higher than the incidence of right-sided cancers in patients undergoing an initial left colectomy. Of 121 patients with two or more large bowel cancers, 56.2% presented with synchronous lesions, while the other 43.8% presented with metachronous (nonsimultaneous) lesions. Of the patients with metachronous large bowel cancer, 28 of 47 had their first lesion on the left side; 13 patients had the first lesion on the right side, while the remaining 6 had the first lesion in the transverse colon. Of the 28 patients who had their first resection on the left side, 12 subsequently developed their second primary carcinoma on the right side. Of the 28 patients who had their first resection on the left side, 13 developed their second primary lesion in the remaining portion of the left side. In contrast, of the 13 patients who had their first resection on the right side of the colon, 10 developed a second lesion in the left side of the colon. Thus, the incidence of second primary carcinomas in patients who first underwent an initial right-sided resection was much higher than the incidence of right-sided cancers in patients undergoing initial left colectomies, and was also higher than the initial incidence of left-sided lesions. These results support the concept that increased delivery of bile acids to the colon may play a role in tumor pathogenesis.

8322 FECAL CONSTITUENTS OF A HIGH-RISK NORTH AMERICAN AND A LOW-RISK FINNISH POPULATION FOR THE DEVELOPMENT OF LARGE BOWEL CANCER. (Eng.) Reddy, B. S.; Hedges, A.; Laakso, K.; Wynder, E. L. (Naylor Dana Inst. for Disease Prevention, American Health Foundation, Valhalla, NY 10595). *Cancer Lett.* 4(4):217-222; 1978.

To elucidate the role of dietary factors in the

etiology of large bowel cancer, dietary composition and feces collected in rural Kuopio, Finland (a low-risk population area for this cancer) and in the New York metropolitan area (a high-risk population area for this cancer) were studied. In both populations, the average daily intake of fat and protein was essentially the same, but the sources of fat came principally from meat in the New York population and from milk and other dairy products in Kuopio. Bile acid and neutral sterol levels and the β-glucuronidase activity of the stool samples was measured. A three-fold greater output of feces ($p<0.05$) was found in Kuopio compared to New York, possibly due to a high intake of fiber-rich cereal products. The fecal bacterial β-glucuronidase activity/mg dry feces was lower in Kuopio than New York (18.6 ± 2.1 versus 69.3 ± 15.8 μg product formed/4 hr), but the total daily output of this enzyme activity was the same for both groups (1,122 ± 158 and 1,545 ± 283 x 10^{-3} μg product formed/day). A significantly lower ($p<0.05$) concentration of secondary bile acids, such as deoxycholic, lithocholic, ursodeoxycholic, and 12-ketolithocholic acids, was found in Kuopio. These bile acids have been found to act as colon tumor promoters in animals. The high fecal bulk noted in Kuopio diluted both the fecal β-glucuronidase activity and the fecal secondary bile acids. The daily excretion of neutral sterols was higher in Kuopio than in New York. The results suggest that diets low in meat and high in fiber coupled with high fecal bulk in Finland may protect against colon cancer by diluting tumorigenic compounds that come in direct contact with the colon mucosa.

8323 THE ROLE OF 5-FLUOROURACIL AS AN ADJUVANT TO THE SURGICAL TREATMENT OF LARGE BOWEL CANCER. (Eng.) Grage, T. B.; Metter, G. E.; Cornell, G. N.; Strawitz, J.; Hill, G. J., III; Frelick, R. W.; *et al.* *In: Adjuvant Therapy of Cancer.* (Amsterdam: Elsevier/North-Holland Biomedical Press): 259-263; 1977.

The effect of adjuvant chemotherapy with 5-fluorouracil (5-FU) upon survival and the recurrence rate was studied in 282 patients with colorectal carcinoma, of whom 204 underwent curative resection and 78 underwent palliative resection. Chemotherapy was started within 30 days after surgery and consisted of i.v. 5-FU (12 mg/kg/day x 4 and then 6 mg/kg on alternate days x 5, followed by 12 mg/kg/week for 1 yr). Follow-up ranged from 3 months to 65 months with a median of 30 months. In the curative group, survival continued to be moderately more favorable in the 5-FU treatment group, although the difference was not significant ($p=0.09$). However, the treatment group had a significantly longer disease-free interval, as determined by plotting a disease-free interval curve, using the Kaplan-Meier life table method ($p=0.045$). The survival and disease-free interval were significantly better for patients with carcinoma of the colon versus rectum ($p=0.038$ and 0.027, resp.). In patients with rectal carcinoma, both survival and disease-free interval were significantly better with chemotherapy ($p=0.024$ and $p=0.037$, resp.). The disease-free interval for patients with lymph node metastases was significantly longer for those on chemotherapy ($p=0.018$). There was no significant difference in survival between the patients who underwent palliative or curative resection. However, the asymptomatic interval in the chemotherapy group was slightly longer ($p=0.027$). It is concluded that the treatment benefit following 5-FU administration as an adjuvant to surgical treatment of colorectal cancer is small, mainly seen in prolongation of the disease-free interval and confined to patients with either lymph node metastasis or patients with rectal carcinoma.

8324 VILLOUS TUMORS OF THE RECTUM AND COLON: DIAGNOSTIC AND THERAPEUTIC PROBLEMS AND STATISTICS ON 78 CASES. (Fre.) Delile, P.; Marche, C.; Edelmann, G. (Hopital Bichat, 170, Bd Ney, 75018 Paris, France). *Ann. Chir.* 31(10): 829-842; 1977.

Among 78 patients with villous tumors of the rectum and colon, the histological diagnosis was purely villous tumors in 59, villous polyadenomas in 11, and adenocarcinomas with villous processes in 8. The purely villous tumors are defined as those consisting only of epithelium; however, they can be sessile (45), nonprotruding (6), or in the form of polyps (8). Villous polyadenomas consist of benign tumors of glandular and epithelial tissue. Excluding the villous adenocarcinomas, there were 31 benign villous tumors and 7 suspect and 32 definitely malignant tumors. All the villous tumors of the colon and rectosigmoid (21) whether malignant or benign, were resected by the abdominal route, and there were no postoperative complications. A recall period of 6 months to 17 yr on these patients revealed two lost to follow-up and no recurrences, even though six patients had had stage II, III, or IV tumors. Various surgical procedures were used to extirpate 34 villous tumors of the rectum (21 benign, 13 malignant) implanted within 12 cm of the anal margin: transanal tumorectomy (27, of which 12 required further surgery or electrocoagulation for recurrence), trans-sacral tumorectomy (3), abdomino-transanal amputation of the rectum (2), and abdominoperineal amputation of the rectum (2), with 3 operations for recurrence. The recurrences always followed incomplete excision. It is stated that villous tumors of the rectum can always be treated by extensive local excision rather than rectal amputation unless there is obvious invasion of the submucosa or tumor degeneration.

8325 AGE-RELATED CHANGES IN THE COLONIC BLOOD SUPPLY: THEIR RELEVANCE TO ISCHAEMIC COLITIS. (Eng.) Binns, J. C.; Isaacson, P. (Southampton Univ. Hosp., Southampton SO9 4XY, England). *Gut* 19(5):384-390; 1978.

The anatomy of the colonic circulation was studied

by performing postmortem angiograms in 37 subjects of different ages (neonate-92 yr) to determine if age-related changes in the colonic blood supply are relevant to ischemic colitis due to nonocclusive mesenteric ischemia. Radiopaque material was injected into the superior mesenteric artery (SMA) or inferior mesenteric artery (IMA). Good filling of the colonic circulation was obtained in 23 of the 29 cases in which the SMA was injected. Of 13 cases of occlusion or severe stenosis of the IMA ostium, complete filling of the colonic circulation was obtained in 9. Good filling of the colonic circulation was obtained in five of the eight cases in which the IMA was injected. The diameter of the marginal artery decreased from right to left. The average diameters of the artery at the ascending colon, transverse colon, splenic flexure, and descending colon were 1.6 mm, 1.13 mm, 0.94 mm, and 0.89 mm, resp. In adults, the diameters were constant over the wide range of ages. In 17 of the 37 colons, the artery was continuous and specifically, no interruption or unusual narrowing was seen at the splenic flexure. In 11 other colons, the artery had been damaged during removal of the colon but appeared to be continuous. In young subjects, the long colic arteries were straight with almost complete absence of tortuosity. Regression lines calculated from graphs plotting tortuosity against age indicated that tortuosity of these vessels increases significantly with age ($p<0.01$ in transverse colon, $p<0.02$ at splenic flexure, $p<0.05$ in ascending colon, and $p<0.01$ in descending colon). These results demonstrate a distinct age-related tortuosity of the long colic arteries, which could account for the increasing incidence of ischemic colitis with age. No anatomical basis for the higher incidence of left-sided involvement was found, and no critical point in the arterial circulation at the splenic flexure was demonstrated.

8326 MANOMETRIC INVESTIGATION OF THE ANAL CANAL IN PRIMARY CHRONIC FISSURE BEFORE AND AFTER DILATATION OR SPHINCTEROTOMY. (Ger.) Fischer, M.; Thermann, M.; Hamelmann, H. (Chirurgische Universitatsklinik, Robert-Koch-Strasse 8, D-3550 Marburg, W. Germany). *Chirurg* 49(2): 111-113; 1978.

Manometric investigations were performed in the anal canal before and 6 months after stretching or sphincterotomy in 28 patients with primary chronic fissure of the anal canal. Compared with a proctologically healthy control group and with the values measured before intervention, the length of the functional anal canal did not change significantly after stretching and sphincterotomy, but the position of the maximum resting pressure shifted orad, though remaining within 1-2 cm from the anocutaneous border. The maximum resting pressure in the anal canal was significantly higher during the chronic fissure than either after stretching and sphincterotomy ($p<0.001$) or in the controls but there was no significant difference from the controls after the intervention. The findings indicate that an elevated resting anal pressure is one of the pathogenetic mechanisms in the development of primary chronic fissure and that it is eliminated efficiently by stretching or sphincterotomy.

8327 ULTRASTRUCTURE OF THE "TRANSITIONAL" MUCOSA ADJACENT TO LARGE BOWEL CARCINOMA [Abstract]. (Eng.) Riddell, R. H.; Levin, B. (Univ. Chicago, Chicago, IL). *Gastroenterology* 72(5, Part 2):1119; 1977.

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8332 SEQUENTIAL ANTITUMOR IMMUNOREACTIVITY AND CARCINOEMBRYONIC ANTIGEN LEVELS AS A GUIDE TO PROGNOSIS IN COLORECTAL CARCINOMA. (Eng.) Nairn, R. C.; Nind, A. P. P.; Pihl, E.; Ceredig, R.; Jablonka, I. M.; Cuthbertson, A. M.; *et al.* *Aust. N.Z. J. Surg.* 47(5):637-641; 1977.

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8374 HIRSCHSPRUNG'S DISEASE: AN APPRAISAL OF HISTOCHEMICALLY DEMONSTRATED ACETYLCHOLINESTERASE ACTIVITY IN SUCTION RECTAL BIOPSY SPECIMENS AS AN AID TO DIAGNOSIS. (Eng.) Lake, B. D.; Puri, P.; Nixon, H. H.; Claireaux, A. E. (Hosp. Sick Children, Great Ormond St., London WC1N 3JH, England). *Arch. Pathol. Lab. Med.* 102(5):244-247; 1978.

8375 FIBEROPTIC ENDOSCOPY OF THE GASTROINTESTINAL TRACT IN INFANTS AND CHILDREN: II. FIBEROPTIC COLONOSCOPY AND POLYPECTOMY IN 15 CHILDREN. (Eng.) Liebman, W. M. (Univ. California, San Francisco Sch. Medicine, San Francisco, CA 94143). *Am. J. Gastroenterol.* 68(5):452-455; 1977.

8376 HEMORRHAGE FROM THE LARGE BOWEL WITH SPECIAL REFERENCE TO ANGIODYSPLASIA AND DIVERTICULAR DISEASE. (Eng.) Welch, C. E.; Athanasoulis, C. A.; Galdabini, J. J. (Massachusetts General Hosp., Boston, MA). *World J. Surg.* 2(1): 73-83; 1978.

8377 IMPERFORATE ANUS AND COLON CALCIFICATION IN ASSOCIATION WITH THE PRUNE BELLY SYNDROME. (Eng.) Morgan, C. L., Jr.; Grossman, H.; Novak, R. (Duke Univ. Medical Center, Box 3834, Durham, NC 27710). *Pediatr. Radiol.* 7(1):19-21; 1978.

8378 A TRIAL OF TRIMEBUTINE IN SPASTIC COLON. (Eng.) Luttecke, K. (No affiliation given). *J. Int. Med. Res.* 6(2):86-88; 1978.

8379 PROPHYLAXIS OF ADHESIONS WITH TRASYLOL IN CASES OF PERFORATED APPENDICITIS IN CHILDREN. (Eng.) Perovic, S.; Maksimovic, L. J.; Djaja, M.; Tonic, L. T. (Pediatric Surgical Clinic, Belgrade, Yugoslavia). *J. Int. Med. Res.* 6(2):89-93; 1978.

8380 URINARY TRACT COMPLICATIONS WITH RECTAL SURGERY. (Eng.) Beahrs, J. R.; Beahrs, O. H.; Beahrs, M. M.; Leary, F. J. (Mayo Clinic, 200 First St. SW, Rochester, MN 55901). *Ann. Surg.* 187(5):542-548; 1978.

See also, 7979, 8128, 8140, 8147, 8148, 8150, 8160, 8166, 8169, 8180, 8200, 8201, 8248, 8287, 8382, 8386, 8395, 8403, 8409, 8439, 8564, 8566, 8572, 8575, 8596, 8601, 8602, 8603, 8606.

LARGE INTESTINE

Ulcerative Colitis

8381 A RADIOLOGICAL AND PATHOLOGICAL CORRELATION OF THE MUCOSAL CHANGES IN ULCERATIVE COLITIS. (Eng.) Bartram, C. I.; Walmsley, K. (St. Mark's Hosp., City Rd., London EC1V 2PS, England). *Clin. Radiol.* 29(3):323-328; 1978.

The results of double-contrast barium enema (DCBE) in 37 patients with ulcerative colitis were compared with the pathological appearance of resected colonic specimens from each of these patients to assess the value of DCBE in this disease. DCBE was carried out without bowel preparation in 29 patients. Total colitis was demonstrated macroscopically in 26 resected specimens and histologically in 32. Radiological and pathological agreement of the extent of the disease was present in 12 patients with total colitis and in 5 patients with subtotal colitis. In 20 patients, total colitis was diagnosed histologically, but radiologically the disease extended proximally to the hepatic flexure in 8, to the transverse colon in 6, to the splenic flexure in 4, and to the descending colon in 2. The radiological appearance was placed into one of the following categories by two observers: granular fine (GF), granular coarse (GC), coarse surface irregularity (CSI), and mucosal excrescences (ME). The pathological appearance was graded according to the following categories: type 1, rough granular surface with no obvious defects; type 2, a similar texture but with small surface defects; type 3, severe disruption of the surface due to ulceration; and type 4, the presence of mucosal tags. Observer agreement was present in 74% of the cases classified as GC and in 86% of cases classified as CSI; agreement was poor with GF and ME. Where observer agreement was present, the radiological and pathological classifications were well correlated. GF was associated with type 1 pathological appearance in 75%, GC with type 2 in 74%, and CSI with type 3 in 85%. With ME the numbers were too small for a valid conclusion. With qualifications, the DCBE without bowel preparation provides useful information as to the extent and severity of ulcerative colitis.

8382 LEUCOCYTE MIGRATION TEST WITH AUTOLOGOUS COLONIC MUCOSA AS ANTIGEN IN PATIENTS WITH ULCERATIVE COLITIS. (Eng.) Astrup, L.; Rasmussen, S. N.; Binder, V. (Frederiksberg Hosp., Ndr. Fasanvej 59, 2000 Copenhagen F, Denmark). *Scand. J. Gastroenterol.* 12(8):951-955; 1977.

The immunological parameters of ulcerative colitis (UC) were evaluated by the leukocyte migration agarose technique (LMAT, Clausen's technique) using the patient's own colonic mucosa as a possible anti-

gen. To determine if a possible autohypersensitivity was specific to colonic mucosa, the results were compared with those obtained using autologous skin and buccal mucosa. The migration of peripheral WBC was studied in 16 untreated UC patients (5 inactive, 7 moderately active, 4 very active). Twelve patients with peptic ulcer, irritable colon, or hemorrhoidal tumors served as controls. The final concentration of protein in the three types of antigen solution was standardized to 100 µg/ml medium. The final concentration of WBC in the test solutions was 2.8×10^8/ml. The migration index (MI, ratio of area with antigen to the area without antigen) for UC patients and for controls was 1.02 ± 0.08 and 1.03 ± 0.13, resp., with rectal mucosa as antigen, 1.04 ± 0.08 and 1.07 ± 0.14, resp., with skin as antigen, and 0.98 ± 0.10 and 1.02 ± 0.09, resp., with buccal mucosa as antigen. The MI was considered to be within normal limits in all patients, regardless of the antigen used. No significant differences were demonstrated among the MI values. No correlation between the MI and the clinical activity of UC was demonstrated. The authors conclude that no antigen activity of autologous colon mucosa, buccal mucosa, or skin was demonstrable in UC by means of the LMAT but question whether the concentration of antigen used in the study was sufficient.

8383 FLUORESCENT ANTI-COLONIC AND E. coli ANTIBODIES IN ULCERATIVE COLITIS. (Eng.) Marcussen, H. (Bispebjerg Hosp., Bispebjerg Bakke, 2400 NV, Copenhagen, Denmark). *Scand. J. Gastroenterol.* 13(3):278-281; 1978.

To examine the possibility that *Escherichia coli* lipopolysaccharide (LPS) cross-reacting antigens induce autoimmunization to colon antigens in patients with ulcerative colitis (UC), the antigenic similarities between *E. coli* and colon mucosa from guenons and from rats were investigated and several *E. coli* serotypes were compared with each other antigenically using an indirect immunofluorescence technique. Sera from eight patients with UC yielded positive staining of goblet cells (4 with both antigens, 2 with guenon antigen only, and 2 with rat antigen only); sera from two patients were entirely negative, as were sera from five hospital controls. When the 10 patient sera were applied to agar preparations of 13 *E. coli* O group strains, there was 1 reaction with the O 1 serotype, 4 with the O 2 serotype, 3 with the O 8 serotype, and 5 with the O 14 serotype; there were 13 doubtful reactions between patient sera and 8 other *E. coli* O group serotypes. The only reactions among the control sera were two doubtful reactions, one each to serotypes O 8 and O 25. The fluorescent-staining activity of the seven patient sera that had reacted with *E. coli* antigens O 2, O 8, and O 14 was abolished by absorption with sediments of the corresponding bacteria. The strength of the immunofluorescence reaction was found to decrease as the sera were progressively diluted from 1:2 to 1:32. The results indicate that antigens from colon mucosa share antigenic determinants with some *E. coli* bacteria or bacterial components (possibly LPS).

8384 IgG MYELOMA PRESENTING AS ULCERATIVE COLITIS. (Eng.) Haeney, M. R.; Ross, I. N.; Thompson, R. A.; Asquith, P. (East Birmingham Hosp., Birmingham B9 5ST, England). *J. Clin. Pathol.* 30(9):862-867; 1977.

To document the association of ulcerative colitis and myeloma, the cases of two elderly patients (a 63-yr-old man and a 61-yr-old woman) who presented with profuse bloody diarrhea and weight loss are discussed. Sigmoidoscopy revealed a uniformly edematous, hyperemic rectal mucosa with mucus and contact bleeding in the man and a granular, edematous rectal mucosa with mucopus and severe contact bleeding in the woman. Barium enema revealed loss of sigmoidal mucosal pattern, poorly distensible rectum, and increased rectosigmoid space in both patients. Rectal biopsies showed nonspecific proctitis, infiltration by lymphocytes and polymorphonuclear leukocytes, and morphologically normal plasma cells in the man and active nonspecific proctitis, increased cellularity and polymorphonuclear leukocytes in the lamina propria, early crypt abscess formation, and no evidence of amyloid in the woman. Bone marrow histology in the man revealed atypical plasma cells showing nuclear immaturity of which 18-20% were IgG-staining and <1% were IgA- or IgM-staining by immunofluorescence (IF), and in the woman it showed atypical plasma cells forming clumps of which 10% were IgG-staining by IF; the woman's plasma cells were multinucleate and pleomorphic with prominent nucleoli. Both patients had a monoclonal IgG serum paraprotein with free light chains (kappa in the man, lambda in the woman) in the urine. Both patients were treated with salazopyrine and steroid retention enema for the ulcerative colitis. Cyclophosphamide and prednisolone therapy (varying doses)was initiated in the male patient, and this therapy led to an improvement in both gastrointestinal disease and myeloma. Specific treatment for the myeloma was withheld in the second patient. Because the etiology of ulcerative colitis is unknown, it is difficult to assess the relation between it and IgG myeloma; the decreased resistance to infection that occurs during IgG myeloma may increase the potential for invasion by hypothetical microorganisms. It is suggested that a diagnosis of myelomatosis should be considered in elderly patients presenting with ulcerative colitis.

8385 A CONTROLLED COMPARISON OF CORTICOTROPIN AND HYDROCORTISONE IN TREATMENT OF SEVERE PROCTOCOLITIS. (Eng.) Powell-Tuck, J.; Bucknell, N. A.; Lennard-Jones, J. E. (St. Mark's Hosp., City Rd., London EC1V 2PS, England). *Scand. J. Gastroenterol.* 12(8):971-975; 1977.

The effectiveness of corticotropin (ACTH) and hydrocortisone in the treatment of severe proctocolitis was compared using parenteral doses of the drugs arranged to give similar blood cortisol levels.

Sixteen patients with confirmed proctocolitis were studied in a 7-day trial. Seven patients were treated with one daily i.m. injection of 80 U of ACTH gel. Nine patients received a constant infusion of hydrocortisone hemisuccinate, 400 mg/24 hr. Three patients receiving hydrocortisone required a reduction of dose because of side-effects. The two groups were comparable regarding the extent, severity, and previous treatment of the disease. Ten patients had received prior treatment with oral steroids immediately before entering the study (hydrocortisone, 6; ACTH, 4). Nine patients were discharged on medical treatment (hydrocortisone, 6; ACTH, 3), one had elective surgery after a good response to hydrocortisone treatment, and six required colectomy after showing no improvement on medical treatment (hydrocortisone, 2; ACTH, 4). The mean length of hospital stay was 30 days in the hydrocortisone group and 40 days in the ACTH group. The measured plasma cortisol levels were comparable except immediately prior to ACTH injection, when the levels in the ACTH patients were significantly ($p<0.01$) lower than in the hydrocortisone patients. No correlation was found between the plasma cortisol levels and the observed therapeutic response. The higher incidence of side-effects with hydrocortisone treatment appeared to be related to the high sustained cortisol levels over the 24-hr period. Patients who had received prior treatment with prednisolone immediately before entering the study had a lower response rate (5/10) to medical treatment than did those who had not been previously treated (5/6). Although the results do not suggest a major advantage for either treatment, the trend favors corticosteroid therapy over ACTH in the treatment of acute colitis. The parenteral administration of a synthetic glucocorticoid such as prednisolone (60 mg equivalent to 300 mg of hydrocortisone) may be the treatment of choice.

8386 INFLAMMATORY BOWEL DISEASE (ULCERATIVE COLITIS). (Eng.) Alpers, D.; Avioli, L. V. (Jewish Hosp. of Saint Louis, Saint Louis, MO). *Arch. Intern. Med.* 138(2):286-291; 1978.

8387 SEQUENTIAL PURE RED CELL AND MEGAKARYOCYTE APLASIA ASSOCIATED WITH CHRONIC LIVER DISEASE AND ULCERATIVE COLITIS. (Eng.) Fox, R. M.; Firkin, F. C. (Ludwig Inst. Cancer Res., Blackburn Building, Univ. Sydney, Sydney, New South Wales, 2006, Australia). *Am. J. Hematol.* 4(1):79-85; 1978.

8388 ULCERATIVE AND GRANULOMATOUS COLITIS. (Eng.) Sommers, S. C. (Lenox Hill Hosp., New York, NY 10021). *Am. J. Roentgenol.* 130(5):817-823; 1978.

8389 THE SURGICAL ASPECTS OF CHRONIC MUCOSAL INFLAMMATORY BOWEL DISEASE (CHRONIC ULCERATIVE COLITIS). (Eng.) Van Heerden, J. A.; McIlrath, D. C.; Adson, M. A. (Mayo Clinic, 200 First St. SW, Rochester, MN 55901). *Ann. Surg.* 187(5):536-541; 1978.

8390 INFANTILE FACTORS IN TWO CASES OF ULCERATIVE COLITIS. (Eng.) Levitan, H. L. (Queen Elizabeth Hosp., Montreal, Quebec, Canada). *Int. J. Psychiatry Med.* 8(2):185-190; 1977/1978.

8391 INFLAMMATORY BOWEL DISEASE WITH SOME COMMENTS ON ULCERATIVE COLITIS. (Eng.) Cooke, W. T. (General Hosp., Birmingham, England). *Rend. Gastroenterol.* 9(3):218-227; 1977.

8392 IMMUNOLOGICAL ASPECTS OF ULCERATIVE COLITIS. (Spa.) Herrerias, J. M.; Perez Ruilopez, M. A.; Pellicer, F. J.; Olivan, J.; Sanchez Cousteau, J. A.; Garrido, M. (Hospital Universitario de Sevilla, Seville, Spain). *Rev. Esp. Enferm. Apar. Dig.* 50(4):409-418; 1977.

8393 IMMUNOLOGICAL ASPECTS OF ULCERATIVE RECTOCOLITIS. (Ita.) Luzi, G.; Pandolfi, F.; Paganelli, R.; Aiuti, F. (Istituto di Clinica Medica Generale e Terapia Medica III, Universita degli Studi, Rome, Italy). *Recent. Prog. Med. (Roma)* 63(3):213-227; 1977.

8394 IMMUNOPATHOGENESIS OF ULCERATIVE COLITIS. (Ger.) Gebbers, J. O.; Otto, H. F. (Institut fur Pathologie der Universitat, 2000 Hamburg 20, Martinistr. 52, W. Germany). *Dtsch. Med. Wochenschr.* 102(11):400-406; 1977.

8395 CHANGES IN FLUID-ELECTROLYTE METABOLISM IN INFLAMMATORY DISEASES OF THE LARGE INTESTINE. (Rus.) Oleinikov, P. N.; Levitan, M. Kh.; Ilysheva, G. N.; Brandina, A. M.; Kataeva, L. G. (Scientific Res. Lab. and Clinic Proctology, RSFSR Ministry of Public Health, Moscow, USSR). *Klin. Med. (Mosk.)* 55(2):61-66; 1977.

See also, 8140, 8143, 8167, 8180, 8266, 8304, 8348, 8349, 8376.

8396 THE ROLE OF CAERULEIN IN TESTS OF EXOCRINE PANCREATIC FUNCTION. (Eng.) Cavallini, G.; Mirachian, R.; Angelini, G.; Vantini, I.; Vaona, B.; Bovo, P.; *et al.* (Clinica Medica III, Policlinico di Borgo Roma, Verona, Italy). *Scand. J. Gastroenterol.* 13(1):3-15; 1978.

The results of exocrine pancreatic function tests using rapid i.v. injection of secretin (1 clinical U/kg) plus cerulein (100 ng/kg) or cholecystokinin (1 or 2 Ivy Dog U/kg) were compared with those in which a continuous infusion was used. Sixty-three controls and 69 patients with chronic pancreatitis were placed in one of the above groups. Duodenal juice was collected for two and four 30-min periods in the subjects receiving the i.v. injection and the infusion, resp. Significant differences in volume secretion and bicarbonate and enzyme secretion were found in the patients as opposed to the controls at 30 min following the rapid i.v. injection of either secretin plus cerulein or secretin plus cholecystokinin. However, pancreatic stimulation was submaximal, and there was marked overlap between individual values of the parameters of pancreatic function in the patients and controls. Side-effects (diarrhea, upper right abdominal pain, and flushing) were noted in four patients who received secretin plus cerulein by i.v. injection. Better differentiation between normal and abnormal pancreatic function was provided by the continuous infusion of secretin and cerulein or secretin and cholecystokinin; the infusion of either combination produced similar results except in the case of volume. No side-effects were observed.

8397 PANCREATIC ASCITES. (Fre.) Lienhard, P.; Hay, J. M.; Maillard, J. N. (Hopital Louis-Mourier, 92701 Colombes Cedex, France). *Chirurgie* 28(5):379-380, 383-384, 387-390; 1978.

The diagnosis and treatment of pancreatic ascites (defined as massive and recurring ascites occurring in the course of an isolated chronic pancreatitis) are discussed, and six cases are reported. Four of the patients had pseudocysts of the pancreas, and fluid was escaping into the peritoneal cavity as a result of rupture of the cyst. The pseudocysts were usually secondary to chronic pancreatitis in alcoholics (3/4 patients in this series). Two of these patients were treated by cystogastrotomy and two by splenopancreatectomy; a death caused by acute respiratory insufficiency 8 days postoperatively was the only treatment failure. The fifth patient had a small fistula on the head of the pancreas that developed after a partial gastrectomy for a duodenal ulcer; the pancreatic ascites was cured by insertion of an external drain (case does not fit the definition). The sixth patient had chronic pancreatitis attributed to alcoholism; however, no communication between the pancreas and the peritoneal cavity was seen at exploratory laparotomy. This patient did not have a recurrence of the ascites after the peritoneal cavity was drained a second time. The presence of chronic pancreatitis is not known in about 25% of patients with pancreatic ascites. Definitive diagnosis can be made by x-ray identification of the pseudocyst, presence of high levels of amylase in the ascitic fluid, and presence of bisalbumin in serum and ascitic fluid. Other treatment besides surgery may be the use of diuretics, steroids, antienzymes, and radiotherapy.

8398 ANGIOGRAPHIC EVALUATION OF GASTROINTESTINAL HAEMORRHAGE COMPLICATING PANCREATIC DISEASE. (Eng.) Cosgrove, H.; Legge, D.; O'Connell, F. X.; Weir, D. (Mater Hosp., Dublin, Ireland). *Clin. Radiol.* 29(3):289-293; 1978.

The role of angiography in the management of gastrointestinal hemorrhage is discussed in relation to seven patients with gastroesophageal varices due to splenic or portal vein obstruction. Angiographic studies included celiac, superior mesenteric, splenic, and left gastric injections. In all cases, angiographic findings were confirmed at operation or at post-mortem examination. Five of the patients had pancreatitis and two had carcinoma. All but two of them had developed complications of pancreatic disease including pseudocyst, abscess, biliary obstruction, or pancreatic insufficiency. Such complications present a risk for the development of splenoportal hypertension and variceal bleeding. Five patients had occlusion or compression of the splenic vein and two had portal vein occlusion. Both patients with portal vein obstruction and two of those with splenic vein obstruction experienced acute and massive gastrointestinal bleeding. In three cases the bleeding resulted from the varices, and in one case, it resulted from tumor invasion into the duodenum. Endoscopic examination of two patients with splenic vein occlusion showed blood in the gastroesophageal region and excluded other causes of hemorrhage. One patient died; the other had a splenectomy based on angiographic demonstration of the source of the bleeding, and no further bleeding was experienced. In a patient with portal vein obstruction, endoscopy revealed blood in the pylorus of the stomach, and angiography showed that the source of the bleeding was the transverse pancreatic artery in the region of the head of the pancreas, due to mucosal erosion from carcinoma. The varices had not bled. Surgical resection of the duodenum and part of the tumor prevented further hemorrhage. The authors conclude that angiographic evaluation may be indicated in any patient with established or complicated pancreatic disease and that selective visceral angiography is essential for the proper management of these patients when gastrointestinal hemorrhage occurs.

8399 EVALUATION AND FOLLOW-UP OF PANCREATIC ARTERIOGRAMS. A NEW ROLE FOR ANGIOGRAPHY IN THE DIAGNOSIS OF CARCINOMA OF THE PANCREAS. (Eng.) Herlinger, H.; Finlay, D. B. L. (St. James' Univ. Hosp., Leeds, England). *Clin. Radiol.* 29(3): 277-284; 1978.

The use of pancreatic angiography in the diagnosis

of pancreatic carcinoma was evaluated by examining arteriograms for 103 patients (35 of whom had carcinoma). There were three false-negative and seven false-positive diagnoses of carcinoma, giving an overall accuracy rate of 90.3%. After reviewing the films retrospectively, only one false-negative and one false-positive case could not be identified. Among the five periampullary carcinomas, the arteriogram in one case presented as a small avascular area with only slight serpiginous encasement of surrounding small vessels; another showed smooth encasement and displacement of the gastroduodenal artery without any other feature of carcinoma. In two other patients, the angiographic presentation was similar to that seen in the cases of pancreatic carcinoma. Pathological vessels were seen in at least part of the tumor in all 30 cases of pancreatic carcinoma. Two carcinomas showed intense tumor blush. Serrated encasement occurred in 61% of the 30 pancreatic carcinomas, smooth encasement in 22%, and serpiginous encasement in 17%. The smallest lesions detected by pancreatic arteriography were less than 2 cm in diameter; among these, one was found in the ampulla, one in the head/body region, and two in the tail of the pancreas. Since noninvasive imaging techniques are now available, angiographic diagnosis of pancreatic carcinoma is often not required. The new purpose of angiography in the diagnosis of pancreatic carcinoma is the demonstration of small and still curable lesions.

8400 THE PANCREAS AND IMMUNOGLOBULINS. III. SECRETORY LEVELS OF IMMUNOGLOBULINS G, A, M IN PATIENTS WITH HYPERSECRETORY DISEASE, POST-KIDNEY TRANSPLANTATION AND PANCREATIC CARCINOMA. A NEW DIAGNOSTIC TEST. (Eng.) Soto, J. M.; Aufses, A. H., Jr.; Dreiling, D. A. (Mt. Sinai Sch. Medicine, City Univ. New York, 100th St. and 5th Ave., New York, NY 10029). *Am. J. Gastroenterol.* 68(1):34-37; 1977.

The IgG, IgA, and IgM levels in pancreatic juice were investigated in three groups of patients to determine the diagnostic value of this parameter. Renal transplant patients (4) on immunosuppressive drugs, patients with hypersecretory disease (7 with minimal pancreatitis and 7 with hypersecretory pancreatic dysfunction of varying etiology), and 9 patients with pancreatic carcinoma were tested and compared with 38 normal patients and 15 patients with chronic pancreatitis. There were no significant differences between the normal controls and either the renal transplant patients or the patients with hypersecretory disease. In patients with pancreatic carcinoma, IgA was absent from pancreatic juice except for high levels in patients with very early lesions and from whom juice was obtained directly from the duct of Wirsung following secretin stimulation; the data for IgG levels in these patients showed scatter, but the average level was markedly higher than in any other group. The IgM level in carcinoma patients was not different from that in any other group. It is concluded that immunosuppressive therapy does not alter the immunoglobulin content of pancreatic juice, that hypersecretion does not dilute pancreatic immunoglobulin concentrations, and that the altered immunoglobulin profile in pancreatic carcinoma patients may constitute a new diagnostic test for pancreatic cancer.

8401 E.R.C.P. IN PANCREATIC DISEASE [Abstract]. (Eng.) Hadfield, A. (London Hosp., London, England). *Br. J. Radiol.* 51(601):66; 1978.

8402 PANCREATIC SCANNING [Abstract]. (Eng.) Testa, H. J.; Prescott, M.; Braganza, J. (Royal Infirmary, Manchester, England). *Br. J. Radiol.* 51(601):66; 1978.

8403 SUBCLINICAL RAT SOLUBLE VITAMIN DEFICIENCY IN TREATED PATIENTS WITH PANCREATIC INSUFFICIENCY [Abstract]. (Eng.) Dutta, S. K.; Costa, B. S.; Russell, R. M. (Veterans Admin. Hosp., Baltimore, MD). *Am. J. Clin. Nutr.* 31(4):707; 1978.

8404 SPLENIC EROSION AND HEMORRHAGE SECONDARY TO PANCREATIC PSEUDOCYST: A REVIEW OF THE LITERATURE AND ADDITIONAL CASE REPORT. (Eng.) Shafiroff, B. B.; Berkowitz, D.; Li, J. K.; Fletcher, P. (Montefiore Hosp. and Medical Center, Bronx, NY). *Am. J. Gastroenterol.* 68(2):145-153; 1977.

8405 ENVIRONMENTAL FACTORS AND DISEASES OF THE PANCREAS. (Eng.) Longnecker, D. S. (Dartmouth Medical Sch., Hanover, NH 03755). *Environ. Health Perspect.* 20:105-112; 1977.

8406 CIMETIDINE AS AN ADJUNCT TO ORAL ENZYMES IN THE TREATMENT OF MALABSORPTION DUE TO PANCREATIC INSUFFICIENCY. (Eng.) Regan, P. T.; Malagelada, J. R.; DiMagno, E. P.; Go, V. L. W. (Mayo Clinic, Rochester, MN). *Gastroenterology* 74(2, Part 2):468-469; 1978.

8407 PANEL: CANCER OF THE PANCREAS. (Eng.) Levin, B.; ReMine, W. H.; Hermann, R. E.; Schein, P. S.; Cohn, I., Jr. (Dept. Medicine, Univ. Chicago, Chicago, IL). *Am. J. Surg.* 135(2):185-191; 1978.

8408 A CASE OF HETEROTOPIC PANCREAS IN LYMPH NODE. (Eng.) Murayama, H.; Kikuchi, M.; Imai, T.; Yamamoto, Y.; Iwata, Y. (Fukuoka Univ., Sch. Medicine, 34 Nanakuma, Nishi-ku, Fukuoka 814, Japan). *Virchows Arch. [Pathol. Anat.]* 377(2):175-179; 1978.

8409 CYSTIC FIBROSIS PRESENTING WITH THE MECONIUM PLUG SYNDROME. (Eng.) Rosenstein, B. J. (Johns Hopkins Hosp., Park 2, 601 N. Broadway, Baltimore, MD 21205). *Am. J. Dis. Child.* 132(2):167-169; 1978.

8410 Salmonella typhimurium PANCREATIC ABSCESS: REPORT OF A CASE. (Eng.) Strand, C. L.; Sanders, S. L. (Crawford W. Long Memorial Hosp., Atlanta, GA 30308). *Am. Surg.* 44(3):174-176; 1978.

8411 PANCREATIC ARTERIOGRAPHY [Abstract]. (Eng.) Finlay, D.; Herlinger, H. (St. James' Hosp., Leeds LS9 7TF, England). *Br. J. Radiol.* 51(601):65-66; 1978.

See also, 8128, 8140, 8149, 8154, 8158, 8159, 8167, 8174, 8201, 8305, 8416, 8437, 8533.

PANCREAS

Pancreatitis

8412 STUDY OF THE PRINCIPAL PANCREATIC DUCT BY SONOGRAPHY. (Fre.) Pietri, H.; Palasciano, G.; Aimino, R.; Serafino, X. (Institut J. Paoli I. Calmettes, 232 bd de Sainte Marguerite, F 13273 Marseille Cedex 2, France). *Nouv. Presse Med.* 7(2):113-115; 1978.

A new technique is described for visualizing a dilated main pancreatic duct (Wirsung's canal) by ultrasound. The transducer must be used with a sweep motion in only one direction while positive pressure is exerted on the epigastrium. On a transverse section, the duct appears spindle shaped. It is in the shape of a circle 4 mm to 2 cm in diameter in the center of the pancreas on sagittal section. The method visualized the duct in 4/10 patients with chronic pancreatitis, whereas former methods successfully visualized the duct in 20/140 patients with dilated ducts.

8413 PARENTERAL NUTRITION IN THE TREATMENT OF ACUTE PANCREATITIS: EFFECT ON COMPLICATIONS AND MORTALITY. (Eng.) Goodgame, J. T.; Fischer, J. E. (Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20014). *Ann. Surg.* 186(5):651-658; 1977.

Clinical characteristics of 46 patients with acute pancreatitis treated with total parenteral nutrition (TPN) were examined. The duration of TPN varied from 5 to 90 days, with an average of 28 days. TPN was discontinued after p.o. intake was adequate in 33 of the 46 patients. Five patients were hyperalimented until their death, and TPN was discontinued in two patients when hope for their recovery was lost. TPN was discontinued in six patients because of complications directly related to this treatment modality. Episodes of catheter-related sepsis prompted discontinuation in five patients, while an inability to control rising serum potassium was the reason for discontinuance in the remaining patient. Despite the 15% incidence of acute renal failure, there were only four episodes of metabolic abnormalities that required discontinuation of TPN. Initial glucose intolerance was common (25%), but was usually controlled by temporarily reducing the infusion rate or by incorporating small amounts (10-20 U) of regular insulin to the infusion mixture. There were two episodes of protein intolerance, one occurring in a patient with a portacaval shunt and the other in a patient with persistent bacteremia. Hypocalcemia occurred in seven patients. Twenty-two patients experienced no complications after definitive therapy of their acute attack of pancreatitis, although 18 required surgery. Cardiovascular complications were the most frequent, followed by respiratory insufficiency, renal insufficiency, metabolic abnormalities, surgical complications, and bleeding dyscrasia. The major hyperalimentation-related morbidity involved episodes of catheter sepsis, which occurred in 8 of 99 catheterizations. Nine deaths occurred in this series. Twelve of 15 patients who were critically ill with nonfunctioning gastrointestinal tracts survived, which supports the opinion that the maintenance of nutritional integrity in the absence of a completely functional gastrointestinal tract and in the presence of severe illness is essential. It is concluded that TPN should be seen as a method of support and not as a primary therapy in acute pancreatitis.

8414 GLUCAGON IN THE TREATMENT OF ACUTE PANCREATITIS. (Ita.) Quaranta, L.; Segre, D.; Balestrino, E.; Beltrutti, D. (Ospedale Civile "S. Croce," Cuneo, Italy). *Gas. Med. Ital.* 136(6/7): 369-376; 1977.

A description of acute pancreatitis is given utilizing published accounts, and data on 25 new cases are presented to emphasize the role of glucagon in treating this disease. Patients were given 1 mg of glucagon every 6 hr in continuous infusion in physiological solution. Treatment was continued

for 2-3 days after normalization of serum and urine amylase values. Patients were also given 1,000,000 IU Trasylol/day. There was a rapid decrease in amylasemia in all of the patients. The blood glucose values showed that the patients tolerated the therapy very well. The serum calcium levels oscillated between 8 and 9 mg%. The results, when compared with previous studies in which patients were not treated with glucagon, show that the pharmacological regimen reported in this paper brings about a more rapid remission of the acute phase of the disease, presumably by blocking edema and impeding the development of necrosis and hemorrhage.

8415 PROSPECTIVE STUDIES ON THE SIGNIFICANCE OF PROTEINASE INHIBITORS AND GLUCAGON IN ACUTE PANCREATITIS. (Ger.) Kirsch, A.; Werner, U.; Heinze, D. (Chirurgische Klinik, Stadtisches Krankenhaus im Friedrichshain, DDR-1017 Berlin, Leninallee 49, E. Germany). *Zentralbl. Chir.* 108 (5):291-303; 1978.

The therapeutic effectiveness of glucagon and the proteinase inhibitor Contrykal was studied in a randomized series of 403 patients with acute pancreatitis. All patients received basic therapy consisting of permanent gastric suction, plasma infusion, and analgesia with procaine. Patients receiving Contrykal also received atropine. Mild pancreatitis was diagnosed in 79 cases, medium-degree pancreatitis in 198, and severe pancreatitis in 126. Seventy-five randomly selected patients were treated with Contrykal (60,000-150,000 U) while 75 controls received basic therapy only. There were 10 recurrences, 1 pseudocyst, and 1 psychosis in the control group, while there were 5 recurrences, 1 pseudocyst, 2 psychoses, and 1 pancreatic abscess in the Contrykal-treated group. The average duration of plasma infusion was 60.5 hr without Contrykal and 77.3 hr with Contrykal. The average duration of the hospital stay was 12.8 days without Contrykal and 13.6 days with Contrykal. Seventy-five patients received glucagon (10 mg/day i.v.) in addition to the basic therapy and 75 controls received basic therapy only. In the glucagon-treated group, six patients developed recurrences, four psychoses, three abscesses, and one a pseudocyst. In the control group, five patients developed recurrences, three psychoses, two pseudocysts, and one abscess. The average length of plasma infusion was 72.1 hr in the treated patients versus 65.8 hr in the controls; the average length of the hospital stay was 18.9 days in the treated group versus 14.7 days in the controls. The remaining group of 103 patients received basic therapy only; 2 developed pseudocysts and 2 psychoses. The average duration of hospital treatment was 14.7 days, the average duration of infusion 67.4 hr. The findings suggest that the results were best in the groups receiving the basic therapy only.

8416 INTRASPLENIC PANCREATIC PSEUDOCYST COMPLICATING SEVERE ACUTE PANCREATITIS. (Eng.) Hastings, O. M.; Jain, K. M.; Khademi, M.; Lazaro, E. J. (Martland Hosp., 65 Bergen St., Newark, NH 07107). *Am. J. Gastroenterol.* 69(2):182-186; 1978.

A case of intrasplenic pancreatic pseudocyst complicating severe acute pancreatitis is presented. The patient was a 27-yr-old alcoholic man who presented with a 4-week history of upper abdominal pain, malaise, and dyspnea. The abdomen was distended and tender, and a poorly defined epigastric mass was palpated. A left-sided pleural effusion was present, necessitating thoracocentesis and subsequent thoracostomy. The patient's clinical condition failed to improve with conservative therapy, and exploratory laparotomy was carried out. At celiotomy, a large, thin-walled cyst, involving the distal half of the pancreas and in juxtaposition to the stomach, was encountered. Because of the immaturity of the cyst wall, external drainage was performed. Cyst fluid cultures grew beta hemolytic streptococci. A postoperative upper gastrointestinal series demonstrated extrinsic pressure on the stomach, and a selective celiac arteriogram, carried out 21 days postoperatively, revealed an avascular area of the spleen. At re-exploration 6 weeks after the initial operation, the angiographic avascular area was found to be a cystic mass, which had an amylase content of 733 Conway U. The spleen was enlarged, and splenectomy was performed. Histopathology of the spleen was unremarkable, and the cyst wall showed a non-epithelial lining with chronic inflammatory changes. Early surgical intervention with splenectomy and possibly caudal pancreatectomy is advocated in cases of intrasplenic pancreatic pseudocyst.

8417 ACUTE PANCREATITIS TREATED WITH AND WITHOUT ADDITION OF INSULIN. (Eng.) Hallberg, D. (Karolinska sjukhuset and Serafimerlasarettet, Stockholm, Sweden). *Acta Chir. Scand.* 143(7/8):451-456; 1977.

Two treatment modalities for acute pancreatitis were compared retrospectively by reviewing 365 case histories from a 5-yr period. One modality involved well-supervised conservative treatment with gastric drainage, fluid and electrolytes given i.v., pain sedatives, and anticholinergic drugs; 165 patients were treated 222 times in this way. The second modality added insulin (20-40 IU/1,000 ml of glucose solution, infused i.v. as needed) to the first regimen; 258 patients were treated 408 times in this way. There were 58 patients treated in both ways at different times; 2 of them died while on treatment without insulin. Among 200 patients treated only once with insulin, 44 were treated during a recurrence (none died) and of the other 156, 5 died. Among 107 patients treated without insulin, 10 died and 9 had recurrences among whom 1 died. Thus, the mortality among the 258 subjects treated with insulin at least once was 1.9%, whereas the mortality among the 165 subjects treated without insulin was 7.9% (if the mortality rates are calculated per hospital admission, they are 1.2% among 408 and 5.9% among 272, resp.). These differences are statistically significant ($p<0.05$). There were no other

significant differences between the two groups; age and sex distribution, positive information of gallstone disease or pancreatitis in 0-10 yr after biliary surgery, positive information of alcohol abuse, serum Ca levels, percentage of serious cases, kinds of surgical intervention, or length of hospital stay were the factors examined. The results support a pathogenetic concept for acute pancreatitis that is based on the incorrect activation of cyclic AMP in abdominal organs, leading to enhanced lipolysis, decreased splanchnic blood flow, pain, accumulation of free fatty acids in the splanchnic adipose tissue, hypocalcemia, and fat necrosis in the abdominal adipose tissue. The insulin plus glucose treatment is believed to inhibit or decrease lipolysis and increase splanchnic blood flow and so reverse the postulated series of events that constitute acute pancreatitis.

8418 DISCUSSION: PERSONAL EXPERIENCE WITH ACUTE PANCREATITIS. (Fre.) Leger, L.; Chiche, B.; Ghouti, A. (No affiliation given). *Chirurgie* 103(9):846-847; 1977.

8419 LAPAROSCOPIC STUDY OF THE CHRONIC EVOLUTION OF ACUTE PANCREATITIS. (Ita.) Berengo, A.; de Lalla, F. (Istituto di Malattie Infettive, Universita di Milano, Milan, Italy). *Minerva Dietol. Gastroenterol.* 23(2):103-105; 1977.

8420 ACUTE PANCREATITIS IN PREGNANT WOMEN. (Ukr.) Fedorchenko, V. M. (Kremenchug Obstetrical Ward, Poltava Oblast, Kremenchug, USSR). *Pediatr. Akush. Ginekol.* (4):59-61; 1977.

8421 ACUTE PANCREATITIS AND ITS TREATMENT. (Nor.) Amundsen, N. (Narvik sykehus, Kirurgisk avdeling, Narvik, Norway). *Tidsskr. Nor. Laegeforen.* 97(27):1398-1399; 1977.

8422 TREATMENT OF ACUTE PANCREATITIS. (Fre.) Roy, B.; Pouyat, X. (No affiliation given). *Chirurgie* 103(9):851-853; 1977.

8423 SURGICAL MANAGEMENT OF ACUTE PANCREATITIS. (Fre.) Gautier-Benoit, C.; Hodin, E.; Demonchy (No affiliation given). *Chirurgie* 103(9): 862-863; 1977.

8424 SURGICAL TREATMENT OF ACUTE PANCREATITIS. (Ger.) Heberer, G.; Schildberg, F. W. (Chirurgische Klinik der Universitat, Nussbaumstr. 20, D-8000 Munich 2, W. Germany). *Munch. Med. Wochenschr.* 120(2/3):55-56; 1978.

8425 EARLY SURGERY IN SERIOUS ACUTE PANCREATITIS: REPORT OF A SERIES OF 100 CASES. (Fre.) Mercadier, M. (No affiliation given). *Chirurgie* 103(9):835-845; 1977.

8426 INDICATIONS AND RESULTS OF TOTAL PANCREATECTOMY IN THE TREATMENT OF ACUTE NECROTIZING PANCREATITIS. (Fre.) Alexandre, J. H.; Camilleri, J. P.; Assan, R.; Guerrieri, M. T.; Bonan, A. (Hopital Broussais, 96, rue Didot, 75014 Paris, France). *Chirurgie* 103(9):858-861; 1977.

8427 COMBINATION OF SCINTIGRAPHY AND ULTRASONIC TOMOGRAPHY IN CHRONIC PANCREATITIS. (Ger.) Benz, U. F.; Muller-Brand, J.; Gyr, K. (Universitatsinstitut fur medizinische Radiologie, Spitalstrasse 21, CH-4004 Basel, Switzerland). *Therapiewoche* 27(46):8283-8295, *passim;* 1977.

8428 ELIMINATION OF THE EXOCRINE FUNCTION OF THE PANCREAS IN CHRONIC PANCREATITIS. (Ger.) Mangold, G.; Kummerle, F. (Chirurgische Universitatsklinik, Langenbeckstr. 1, 6500 Mainz, W. Germany). *Dtsch. Med. Wochenschr.* 102(52): 1926-1927; 1977.

8429 DYNAMIC CHANGES IN VARIOUS COMPONENTS OF THE KININ SYSTEM OF THE BLOOD IN CHRONIC PANCREATITIS. (Rus.) Orlov, V. A.; Beliakov, N. V.; Kuznetsov, V. A. (Clinical Hosp. No. 20, Moscow, USSR). *Ter. Arkh.* 49(12):82-84; 1977.

8430 TREATMENT OF PATIENTS WITH CHRONIC PANCREATITIS AT THE ESSENTUKI HEALTH RESORT. (Rus.) Suveiev, I. M.; Babicheva, G. D.; Netesa, E. Ia. (Essentuki Health Resort, USSR Ministry Defense, Essentuki, USSR). *Vrach. Delo* (6):80-82; 1977.

8431 RESECTION PROCEDURE OR SPLANCHNICECTOMY IN CHRONIC PANCREATITIS: INDICATIONS AND COMPARATIVE RESULTS. (Ger.) Vossschulte, K.; Scheld, H. (Chirurgische Universitatsklinik, Klinikstr. 29, D-6300 Giessen, W. Germany). *Munch. Med. Wochenschr.* 119(41):1325-1328; 1977.

8432 PANCREATITIS--A RARE COMPLICATION OF TYPHUS ABDOMINALIS. (Rus.) Borisova, M. A.; Krylova, A. M.; Rybka, I. N.; Prudnikov, B. A.; Solomashchenko, B. S. (Dept. Infectious Diseases, Crimean Medical Inst., Simferopol, USSR). *Vrach. Delo* (9):140-142; 1977.

8433 DIAGNOSTIC AND PHYSIOPATHOLOGIC ASPECTS OF BILIARY PANCREATITIS. (Fre.) Delcourt, A.; Jadot, R. (Rue des Bollandistes 5, 1040, Brussels, Belgium). *Rev. Fr. Gastroenterol.* (132): 68; 1977.

8434 FAMILIAL PANCREATITIS IN HYPERLIPOPROTEINEMIA. (Hun.) Schmidt, I.; Bruncsak, A.; Farago, F. (Bacs-Kiskun megyei Tanacs Korhaza, Kecskemet, Hungary). *Orv. Hetil.* 118(47):2845-2848; 1977.

8435 OSTEOLYTIC LESIONS FOLLOWING TRAUMATIC PANCREATITIS. (Eng.) Neuer, F. S.; Roberts, F. F.; McCarthy, V. (St. Luke's Hosp., Kansas City, MO). *Am. J. Dis. Child.* 131(7):738-740; 1977.

8436 PANCREATIC NECROSIS IN A 3-YEAR-OLD-CHILD. (Rus.) Khodov, S. M. (Dept. Pediatric Surgery, Vladivostok Medical Inst., Vladivostok, USSR). *Vestn. Khir.* 119(7):98-99; 1977.

8437 GASTROSCOPIC DIAGNOSIS OF PANCREATIC PSEUDOCYST RUPTURED INTO THE STOMACH. (Ger.) Bretholz, A.; Frey, P.; Lammli, J.; Corrodi, P.; Knoblauch, M. (Kantonsspital Zurich, Zurich, Switzerland). *Z. Gastroenterol.* 15(10): 638-639; 1977.

See also, 8140, 8154, 8157, 8158, 8170, 8313, 8396, 8397, 8400, 8401, 8405, 8595.

LIVER AND BILIARY TRACT

8438 STUDY OF THE LIVER BY COMPUTERIZED TOMOGRAPHY. EVALUATION OF ONE YEAR'S EXPERIENCE WITH 1,480 INVESTIGATIONS. (Fre.) Osteaux, M.; Coenen, H.; Struyven, J.; Marchal, G.; Huvenne, R.; Jeanmart, L.; *et al.* (Departement de Tomographie Computee, Universite Libre de Bruxelles, 1, rue Heger-Bordet, 1000 Brussels, Belgium). *J. Radiol. Electrol. Med. Nucl.* 58(12):765-772; 1977.

A critical analysis of the use of computerized tomography (Delta Scanner: Ohio Nuclear) in the diagnosis of liver pathology is presented based on study of 1,480 patients. Initially, two studies were performed, one before and one after i.v. injection of contrast product (100 ml methylglucamine diatrizoate). The tomography without contrast was done to avoid masking a hypervascular lesion, but this situation was not encountered, and only the contrast study was done later in the series. The specific pathology for 520 patients was hepatomegaly in 85, cirrhosis in 50, steatosis in 5, abscess in 10, hematoma in 3, ascites in 22, cystic formations in 13, biliary cysts in 7, hepatorenal polycystosis in 2, primary tumor in 16, metastatic infiltrations in 107, regional cancer infiltration of the liver in 4, hepatic artery aneurysm in 1, and gallbladder disease (lithiasis) in 26. There were 12 false-positive results, attributed to images of feeble density. Of the 50 false-negative results, 28 were metastases of small size and 16 were of diffuse pathology, particularly lymphomatosis. Circumscribed pathology, whether of tumoral, infectious, or traumatic origin, was readily visualized. Diffuse lesions, on the other hand, with the exception of hepatic steatosis, were difficult to visualize by computerized tomography.

8439 INVESTIGATIONS ON ALBUMIN EXCRETION OF THE LIVER INTO THE BILE. II. DISC ELECTROPHEROGRAMS OF DUODENAL BILE. (Ger.) Rogos, R.; Petzold, H. (Kreiskrankenhaus, Leipziger Str. 55, DDR-74 Altenburg, E. Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 38(1):27-32; 1978.

Forty-nine samples of A-bile, 45 samples of B-bile, and 60 samples of C-bile from patients with chronic active hepatitis, liver cirrhosis, atrophic gastritis, cholangitis, acute hepatitis, and irritable colon were studied by disc electrophoresis. Protein fractions that migrated to the cathode were found to be predominant. A typical albumin peak was found in 5/60 C-biles only. These findings show a substantial difference from the electropherogram of T-drain bile. The *in vitro* decomposition of albumin from T-drain bile by trypsin suggests that upon entry into the duodenum, the albumin is rapidly and completely decomposed by the excess amount of proteases present and that its decomposition is faster than that of globulins. Since the albumin metabolites are resorbed, this albumin is part of the endogenous protein reserve. The electrophoretic pattern of the duodenal bile proteins is superimposed by that of protein metabolites, and therefore it was not a reproducible diagnostic tool despite the use of a protease inhibitor in the preparation.

8440 TOTAL SERUM BILE SALTS IN LIVER DISEASES. (Ita.) Annoni, G.; Barbi, G.; Del Ninno, E.; Ideo, G. (Istituto di Clinica Medica III dell' Universita di Milano, Milano, Italy). *Minerva Dietol. Gastroenterol.* 23(2):59-65; 1977.

The usefulness of total serum bile salts as an index of liver disease was determined in 117 patients with hepatopathy and possible correlations between this measurement and other common indices of hepatic function were sought. Twelve patients had acute hepatitis, 20 chronic hepatitis, 35 chronic active hepatitis, 35 chronic active hepatitis with cirrhosis, and 15 inactive cirrhosis. All five groups of patients had significantly raised average serum

levels of total serum bile salts in comparison with healthy individuals ($p<0.001$). The coefficient of correlation between bile salt levels and BSP level was highly significant: $p<0.05$ for chronic hepatitis and $p<0.001$ for chronic active hepatitis with or without cirrhosis and for inactive cirrhosis. The results of a meal test showed that the patients with hepatopathy had a notably augmented level of bile salts. The results show that total serum bile salts represent a highly sensitive index of cholestasis and that the meal test best demonstrates the presence of hepatopathy.

8441 DETERMINATION OF HUMAN LIVER CYTOCHROME P-450 BY A MICROMETHOD USING ELECTRON PARAMAGNETIC RESONANCE: STUDY OF 141 LIVER BIOPSIES. (Fre.) Gabrielle, L.; Leterrier, F.; Molinie, C.; Essioux, H.; Cristau, P.; Laverdant, C. (Centre de Recherches du Service de Sante des Armees, 1 bis, rue du Lieutenant-Raoul-Batany, F92141 Clamart, France). *Gastroenterol. Clin. Biol.* 1(10):775-782; 1977.

A micromethod is described for the measurement of the level of cytochrome P-450 in human liver needle biopsies, and the results of such determination in 141 specimens are given. The specimens were obtained from 33 patients with histologically normal livers, 31 patients with bilharziosis but functionally normal livers, 19 with viral hepatitis with symptoms of at least 3 months duration, 3 with extrahepatic cholestasis, 38 with alcohol-induced fatty degeneration of the liver, and 15 with cirrhosis confirmed by histological examination. The method measures the electron spin resonance (ESR) of whole liver samples (3-20 mg) frozen in liquid nitrogen and the amplitude of the P-450 g=2.27 line; it was first tested on liver samples from Sprague-Dawley rats. Calibration of the ESR values for cytochrome P-450 in rat liver was established by comparing the observed values with those obtained by absorption spectrophotometry. The normal value for P-450 level established for patients without liver pathology was 43 ± 20 nmol/g liver. Patients with viral hepatitis with signs of recovery had values of 39.3 ± 21 nmol/g. Patients with persistant or active viral hepatitis had markedly decreased cytochrome P-450 values (4.3 ± 5.7 nmol/g). The patients with fatty liver degeneration had values of 43.9 ± 22.7 nmol/g, whereas the patients with alcoholic cirrhosis had values of 14.7 ± 17.4 nmol/g. The three patients with extrahepatic cholestasis had values of 73.8, 33.3, and 20.5 nmol/g, resp. This method permits determination of the liver enzyme cytochrome P-450 using very small liver samples (3-5 mg) that can be obtained by needle biopsy.

8442 BILE LYSOSOMAL ENZYMES; CHARACTERISTICS AND PATHOLOGICAL SIGNIFICANCE FOR VARIOUS HEPATOBILIARY DISORDERS. (Eng.) Toyoda, S.; Eto, Y.; Aoki, K. (Tokyo Jikei Univ. Sch. Medicine, Nishishinbashi, Minato-ku, Tokyo, Japan). *Clin. Chim. Acta* 79(2):291-298; 1977.

The presence of several glycosidases in human bile was established in 25 patients, and their pathological significance in various hepatobiliary disorders was studied. The activities of β-galactosidase, β-glucosidase, and *N*-acetyl-β-glucosaminidase were demonstrated in the bile of seven normal subjects, using 4-methylumbelliferyl derivatives as substrates. The enzyme activities were increased 60- to 180-fold after the exclusion of bile salts and other small molecular compounds by Sephadex G-50 gel filtration. In patients with cholelithiasis, the mean activities of β-galactosidase and *N*-acetyl-β-glucosaminidase were significantly decreased ($p<0.01$). These activities were also significantly decreased in patients with acute hepatitis and liver cirrhosis ($p<0.01$). β-Galactosidase activity was within normal limits in one patient with gallbladder cancer, and that of β-glucosidase was near the upper limit in patients with bacterial cholangitis. Isoelectric focusing patterns of biliary glycosidases were similar for specimens from patients with hepatobiliary disorders as compared with normal individuals. This investigation presents firm evidence that lysosomal enzyme activities are present in bile.

8443 CHRONIC PATHOLOGY OF THE LIVER IN ITALY. COOPERATIVE RETROSPECTIVE STUDY. (Ita.) Ascione, A.; Barcellona, R.; Benini, G.; Budillon, G.; Candalaresi, M. T.; Cataldi, V.; *et al.* (Associazione Italiana per lo Studio del Fegato, Naples, Italy). *Recent. Prog. Med. (Roma)* 63(5): 447-470; 1977.

The etiology, nosology, and socio-environmental factors of chronic hepatopathies were studied retrospectively and compared to the incidence and location of chronic hepatopathies in four regions of Italy (North, Central, South, and Sicily). Data were collected from 18 hospitals and university clinics on 5,055 cases of chronic hepatopathy observed in 1972-1974. The distribution of the cases was 2,522, 472, 1,614, and 447 in each of the 4 regions, resp. As percentages of the total cases in each region, resp., the incidence of cirrhosis (all types) was 58.3, 67.1, 57.3, and 71.9%; of primary carcinoma was 3.2, 4.2, 3.8, and 10%; of chronic aggressive hepatitis was 6.2, 8.0, 19.1, and 8.5%; of chronic persistent hepatitis was 4.1, 6.1, 5.9, and 3.5%; of noncirrhotic pathology due to alcohol was 21.5, 3.8, 3.1, and 0.7%; and of noncirrhotic pathology secondary to biliopathy was 1.7, 4.6, 5.4, and 0.7%. The differences among all four regions were statistically significant ($p<0.05$) for alcoholic cirrhosis (North>Central>South>Sicily) and for cryptogenic cirrhosis (Sicily>South>Central>North). Posthepatic cirrhosis and chronic aggressive hepatitis were significantly more prevalent in the South ($p<0.05$), and hepatocarcinoma was more prevalent in Sicily ($p<0.05$). Noncirrhotic pathology due to alcohol was significantly more prevalent in the North than in the Central or South regions ($p<0.05$) and significantly more prevalent in the latter two regions than in Sicily ($p<0.05$). When the incidences of various types of cirrhosis are compared with the total number of cases of cirrhosis, significant

($p<0.05$) regional differences were found for cryptogenic cirrhosis (Sicily>South>Central>North), for alcoholic cirrhosis (North>Central>South>Sicily), posthepatic cirrhosis (South>all other regions), and siderochromatosis (North>South, no cases in the other regions). Significant regional differences were found in the distribution of alcoholics and habitual drinkers versus moderate consumers and abstainers ($p<0.05$, North>Central>South>Sicily), in the percentage of female alcoholics (13.1% in the North, $p<0.05$), the percentage of female habitual drinkers (31.4% in the North and 32.2% in the central area, $p<0.01$), in the male/female ratio of the incidence of alcoholic cirrhosis (20.0% in Sicily, $p<0.01$), and in the incidence of primary carcinoma (5.3% in Sicily, $p<0.05$). Patients with hypocaloric diets had greater incidences of chronic hepatopathy than did those with hypercaloric diets. A greater number of people from the lower economic group had cirrhosis due to alcohol, idiopathic siderochromatosis, and noncirrhotic pathology due to alcohol than did those from the middle and upper income groups. Manual workers had a higher incidence of all types of chronic hepatopathy compared with persons in other types of occupations ($p<0.01$). This study is the first overview of the principal problems of chronic liver pathology with respect to geographic regions and of the role of alcohol in this pathology.

8444 LIVER COLLAGEN. (Fre.) Frey, J.; Bayle, J. J. (U.E.R. de Medecine, 30 rue Ferdinand Gambon, F 42100 Saint-Etienne, France). *Lyon Med.* 239(2):87-93; 1978.

Pathological conditions of the liver characterized by fibrosis are reviewed and discussed on the basis of current knowledge of normal collagen structure and metabolism. Three pathogenic mechanisms are considered: (1) modification of gene expression that determines the respective importance of type I and type II collagen, (2) increase in the activity of the enzymes of post-translational biosynthesis, and (3) decrease in collagenolytic activity. Methods of investigating alterations in collagen metabolism that may lead to diagnosis of liver pathology are discussed. These methods measure enzymes or metabolites in the blood and urine that reflect alterations in synthesis or degradation of collagen. The excretion of enzymes involved in collagen biosynthesis such as procollagen-prolylhydroxylase may be increased in hepatomas, and some authors have found an increase in blood and urine hydroxyproline, a collagen component, associated with the hepatic cirrhosis of chronic alcoholism.

8445 ALPHA-FETOPROTEIN: PART ONE OF GENERAL REVIEW. (Fre.) Delpre, G.; Gilat, T. (Hopital Beilinson, Petah Tikva, Israel). *Gastroenterol. Clin. Biol.* 2(1):87-106; 1978.

The literature on various techniques of measuring alpha-fetoprotein (AFP) in different areas of the body (serum, urine, cerebrospinal fluid, saliva) and the pathological conditions associated with high levels of serum AFP are reviewed. One table gives the limits of sensitivity of detection of AFP by 18 different methods, and another table gives the upper, median, and average values of serum AFP in the healthy subject as determined by radioimmunoassay and enzymatic immunoassay. The levels of AFP in the serum associated with primary and metastatic carcinoma of the liver are discussed; variations in AFP have been found in these patients according to age, sex, race, histological type of carcinoma, presence or absence of cirrhosis, size of tumor, and geographical locale of study. AFP levels found in other malignant tumors in adults (including teratoblastomas, embryonic tumors, and gonadal tumors) are discussed with respect to their diagnostic value. Literature on pathogenesis of elevated AFP levels in hepatic and other tumors is reviewed.

8446 GENERAL REVIEW: ALPHA-FETOPROTEIN. PART II. (Fre.) Delpre, G.; Gilat, T. (Beilinson Hosp., Petah Tikva, Israel). *Gastroenterol. Clin. Biol.* 2(2):193-214; 1978.

The literature concerning the involvement of alpha fetoprotein (AFP) in nonmalignant liver diseases (fulminant, acute, and chronic), in hepatocellular carcinoma (HCC) and its diagnosis, in diseases of children and neonates (malignant tumors, liver diseases, and metabolic and genetic diseases), in pregnancy, and in immunity is reviewed. In various nonneoplastic liver diseases such as cirrhosis and hepatitis, the levels of serum AFP have been found to be useful indicators of the extent, progression, and reversal of the process. A definite relationship appears to exist between serum AFP and HBsAg levels in acute and chronic liver diseases that progress to HCC, although there is some controversy about this association and its application in differential diagnosis. There is also some controversy about the usefulness of markedly elevated serum AFP levels as an early indicator of HCC; the response of these AFP levels to chemotherapy and radiotherapy for HCC may be useful in indicating the efficacy of the therapy. In pediatric and neonatal liver diseases there seems to be a gap between the level of serum AFP in normal individuals compared with that in patients as in adults, but the serum AFP level does not aid the differential diagnosis of HCC from hepatitis as it does in adults. Serum AFP levels have been reported to be elevated in children and neonates with primary liver cancer, teratoma, acute viral hepatitis, congenital biliary atresia, in some chronic hepatopathies (such as infantile cirrhosis), and in some genetic and metabolic diseases, but the interpretation of serum AFP levels in these patients must be approached with caution and may depend on the age of the subject. The diagnostic significance of AFP levels in normal and abnormal pregnancies is discussed; the variation of AFP levels in the fetus, in maternal blood, and in amniotic fluid has a normal course, and deviations from these levels may indicate pathologies. AFP in immunity is discussed with respect to individual tolerance to autogenous AFP,

ways in which this tolerance can be disrupted, and the role of AFP in immunosuppression.

8447 ALPHA-FETOPROTEIN IN THE DIAGNOSIS OF MALIGNANT NEOPLASMS. (Ita.) Burke, P.; Bianco, L. (Istituto di Clinica Medica dell' Universita di Torino, Torino, Italy). *Minerva Dietol. Gastroenterol.* 23(2):91-94; 1977.

The incidence of alpha-fetoprotein (AFP) in 42 patients (21 patients had liver cirrhosis, 1 had acute viral hepatitis, 2 had chronic hepatitis, and 18 had various types of cancer) was determined by radial immunodiffusion and a modification of cross-over electrophoresis. Four of the patients had AFP in their sera (2 had primary hepatomas, 1 had metastasis of testicular teratocarcinoma, and 1 had hepatic cirrhosis). The quantitative values for AFP were 310 mg% ml and 230 mg% ml in the cases of primary hepatoma, 1 mg% ml in the case of testicular teratocarcinoma, and >0.7 µg/ml but <1 mg% ml in the case of hepatic cirrhosis. The results show that AFP serum levels are notably greater in cases of hepatoma than in other hepatopathy.

8448 A STUDY OF HEPATOCELLULAR CARCINOMA AMONG JAPANESE FROM THE POINT OF VIEW OF MORPHO-DEVELOPMENTAL PATHOLOGY--GROSS ANATOMICAL TYPES CLASSIFIED IN ITS RELATION TO CAPSULE FORMATION. (Eng.) Nakashima, T.; Sakamoto, K. (Kurume Univ. Sch. Medicine, Kurume, 830, Japan). *Kurume Med. J.* 24(Suppl.)S43-S62; 1977.

One hundred seven necropsies and seven surgically resected livers were studied to develop a system for classifying hepatocellular carcinoma (HCC) with respect to capsule formation. In Japan, encapsulated HCC is frequent (60-70% of the cases), while elsewhere it is infrequent (e.g., 3.6% of the cases in Los Angeles). The classification parameters used were changes in the parenchyma and the growth pattern of the tumor. When classified by changes in the parenchyma, type A tumors are HCC unaccompanied by cirrhosis, and type B tumors are HCC accompanied by cirrhosis. Small HCC's were assigned to a separate class (type C) regardless of the presence or absence of cirrhosis, but in all 13 cases studied, advanced cirrhosis was present. Cirrhosis was present in 84/107 cases, hepatitis B surface antigen (HBsAg) was positive in 47.6% of the cases, and the overall male:female ratio was 6:1. Among type A patients, HBsAg was usually negative, and the sex ratio was 2.3:1. When classified by growth patterns, type I tumors were the infiltrating type (tumor cells infiltrating into the sinusoids and perisinusoidal areas of the adjacent liver parenchyma), type II tumors were the expanding type (tumor tissue forming a mass that expands as the tumor grows, compressing neighboring liver parenchyma that atrophies and disappears and a connective tissue capsule often forming at the boundary--encapsulated type), type III tumors were the multiple type (HCC metastasizing hematogenously at certain stages of growth, forming multiple separate lesions in the liver parenchyma), and type IV tumors were encapsulated HCC having additional infiltrative lesions or encapsulated HCC continuous with infiltrative lesions suggesting a transition. In the present classification, sclerosing HCC was included in the infiltrative type. Other systematic studies of HCC in Japan are described. The present classification was designed to meet the clinical demand for early diagnosis and early surgery in the treatment of HCC.

8449 ULTRASTRUCTURE OF THE LIVER IN HUMAN AMYLOIDOSIS. (Eng.) Eguchi, T.; Fukuda, K.; Okugawa, N.; Tanikawa, K.; Kimura, N. (Kurume Univ. Sch. Medicine, Kurume, 830, Japan). *Kurume Med. J.* 24(4):229-235; 1977.

Liver biopsy specimens from two patients with primary amyloidosis were examined histologically and ultrastructurally. Histological examination revealed that amyloid deposits around vessels in the portal area were minimal in the first patient but extensive in the second. The basic lobular architecture was destroyed as a result of the amyloid deposits in the latter patient. Electron microscopy in the first patient showed minimal development of amyloid fibrils in the space of Disse. Fine amyloid fibrils in this area were arranged in parallel and measured approximately 50-125 A in width. Amyloid material was limited to the portal area. The cytoplasm of the hepatocytes and the bile canaliculus appeared normal. Extracellular spindle-shaped amyloid bundles were seen within invaginations of the plasma membrane of reticuloendothelial cells. Filamentous structures were also noted within the cell, although there was no continuity between extra- and intracellular filaments. In the second patient, hepatocytes appeared to be reduced in proportion to the extent of amyloid deposition. However, the lobular architecture remained normal in some areas, and in these areas a moderate degree of amyloid deposition was noted in the space of Disse. The amyloid deposit within the space of Disse seemed to spread toward the hepatocyte with resultant constriction of the hepatocyte. No amyloid deposits were noted within the hepatocyte, although the endoplasmic reticulum was vesiculated, and other organelles showed moderate changes. Hepatocytes disappeared in the vicinity of massive amyloid deposits. Irregularly shaped reticuloendothelial cells with large nuclei were often found, and dense amyloid deposits were frequently observed between the cytoplasmic processes of these cells. There appeared to be continuity between extra- and intracellular amyloid filaments. It appears that reticuloendothelial cells play an important role in amyloid production.

8450 LIVER DISEASE IN HOMOZYGOUS α_1-ANTITRYPSIN DEFICIENCY. (Ger.) Karitzky, D.; Lesch, R.; Goedde, H. W.; Witt, I.; Boehm, N.; Beckmann, R.; *et al.* (Universitats-Kinderklinik, Mathildenstr. 1, 7800 Freiburg, W. Germany). *Dtsch. Med. Wochenschr.* 103(4):161-166; 1978.

Infantile liver disease was diagnosed in 5 of 12 children with homozygous α_1-antitrypsin deficiency (Pi-type Z). The course of the disease in these patients was extremely variable. One of the five patients died from liver cirrhosis at the age of 14 yr. The clinical, biochemical, and (in 2 cases) the histological findings in the other four patients became normal over a follow-up period of 1-15 yr. The findings indicate that α_1-antitrypsin deficiency, even when associated with liver disease, need not have an unfavorable prognosis.

8451 INTRAHEPATIC CYSTIC BILE DUCT DILATATIONS AND STONE FORMATION: A NEW CASE OF CAROLI'S DISEASE. (Eng.) Mathias, K.; Waldman, D.; Daikeler, G.; Kauffmann, G. (Universitats-Kliniken, Hugstetterstr. 55, D-7800, Freiburg, W. Germany). *Acta Hepatogastroenterol. (Stuttg.)* 25(1):30-34; 1978.

The pathology, clinical signs, diagnosis, differential diagnosis, therapy, and prognosis of Caroli's disease are briefly reviewed, and a new case of the disease is reported. Caroli's disease is a rare familial anomaly with circumscribed saccular dilatations of enlarged intrahepatic bile ducts coated by regular or pseudopapillomatous epithelium and may be combined with additional malformations of the extrahepatic biliary tract as well as with medullary sponge kidney. Infection and stone formation are frequent complications. Most patients complain of upper abdominal pain caused by cholangitis and gallstones; liver abscesses and septicemia may also occur. Patients may therefore present with colic, fever, and icterus. Extrahepatic lithiasis occurs frequently. Diagnosis by i.v. cholangiography (demonstrating the opacification of ectatic bile duct segments) easily overlooks Caroli's disease because the associated lesions are more remarkable; it is improved by tomography. Other diagnostic methods include intra- or postoperative cholangiography, barium studies, endoscopic retrograde cholangiography, and percutaneous transhepatic cholangiography. Laboratory examinations are noncontributory, laparoscopy and laparotomy are unreliable, and liver biopsy is contraindicated. Differentiation between Caroli's disease and biliary cyst formation, destructive cholangitis, biliary obstruction, polycystic liver disease, and solitary hepatic cysts is presented. Surgery is indicated if the disease is limited to one lobe (which should be resected) or if the disease is diffuse (papillotomy or biliodigestive anastomosis may be useful). Gallstone dissolution and prevention and antibiotics are important in symptomatic therapy. Caroli's disease is often life-shortening; patients die of septicemia, cholangitis, liver abscess, cholangiocellular carcinoma, relapsing cholangitis, and pancreatitis. A 31-yr-old woman with a several-year history of upper abdominal pain, colic, and relapsing pancreatitis underwent cholecystectomy and transduodenal papillotomy for cholangiolithiasis, intrahepatic bile duct ectasia, and papillary sclerosis; also, all stones were removed from the choledochal duct. Despite antibiotic therapy for cholangitis and chenodeoxycholic acid therapy (both by infusion through the T-drain and p.o.), cystic dilatations of the bile ducts and intrahepatic gallstones persist.

8452 INTRAHEPATIC PIGMENT STONES: AN ASIAN DISEASE DIAGNOSED WITH RETROGRADE CHOLANGIOGRAPHY. (Eng.) Johnson, L. F.; Walta, D. C. (Walter Reed Army Medical Center, Box 431, Washington, DC 20012). *Am. J. Dig. Dis.* 23(Suppl. 5):13s-18s; 1978.

The case of a patient with intrahepatic biliary pigment stones diagnosed by endoscopic retrograde cholangiography (ERC) is presented, and the authors' experience with this disease is reviewed. The patient, a 29-yr-old Oriental man, presented with recurrent pruritus and jaundice. At the first admission, the SGOT level was 217 IU, and the alkaline phosphatase level was 19.4 Bodansky U. A p.o. cholecystogram showed poor opacification and, at peritoneoscopy, hepatomegaly was noted, with a normal-appearing gallbladder. Liver biopsy showed pericentral cholestasis with triaditis and bridging of portal areas. An i.v. cholangiogram, performed after the resolution of jaundice, revealed normal common and cystic ducts. Hepatic biopsy was complicated by bleeding and prompted surgical intervention, which confirmed the large noncirrhotic liver, normal gallbladder, and normal common bile duct. The biopsy showed bile stasis, portal fibrosis, and mixed inflammatory infiltrate. Two years later, the patient's symptoms recurred, and biopsy revealed features compatible with extrahepatic obstruction. ERC at this time showed an obstruction proximal to the common hepatic duct bifurcation. Intrahepatic biliary radicles did not fill. Ten days later, a percutaneous hepatic cholangiogram failed to opacify any biliary duct despite a demonstrated obstruction on the retrograde cholangiogram. An operative cholangiogram showed a marked impaction of the intrahepatic biliary radicles with fine pigment stones and biliary sludge. A T-tube was placed in the common bile duct, and the patient subsequently received choleretic therapy with phenobarbital and Decholin. Jaundice disappeared, and the serum bile salt concentration dropped to 2.5 mg/100 ml. Fourteen months after the start of choleretic therapy, the biliary radicles were cleared by constant irrigation with a saline-heparin solution. The patient has been asymptomatic on choleretic therapy, although the serum bile salt level increased to 30 mg/100 ml after closing the T-tube tract. In the authors' experience, gallbladder, cystic, and common duct opacification at ERC without intrahepatic filling in the prone, head-down position denotes disease of the intrahepatic biliary radicles.

8453 A COMPUTER SIMULATION STUDY RELATING TO THE TREATMENT OF FULMINANT HEPATIC FAILURE BY HEMOPERFUSION. (Eng.) Berk, P. D. (Liver Unit, DDB:NIAMDD, Bldg. 10, Rm. 4D-52, Natl. Inst. Health, Bethesda, MD 20014). *Proc. Soc. Exp. Biol. Med.* 155(4):535-539; 1977.

Computer simulation techniques were used to investigate what schedule of charcoal hemoperfusion might more rapidly deplete the apparently critical slowly-exchanging extravascular pool of "toxin," which has been hypothesized to accumulate in fulminant hepatic failure and result in liver coma. In contrast to the hypothetical toxin, the internal kinetics of unconjugated bilirubin have been extensively studied, as has its rate of clearance from blood during hemoperfusion with an albumin-agarose "artificial liver" device. In addition, unconjugated bilirubin has an extrahepatic, extravascular pool that exchanges slowly with plasma at a rate not inconsistent with that which must also be obtained for the hypothetical toxin of fulminant hepatic failure. Accordingly, the effects of various albumin-agarose hemoperfusion treatment schedules on the rate of depletion of the extrahepatic extravascular pool of unconjugated albumin were studied. The extrahepatic, extravascular bilirubin pool in an 18-yr-old woman with type I Crigler-Najjar syndrome decreased by only 15% by the end of a 4-hr hemoperfusion period. Bilirubin content continued to fall after the completion of the perfusion; a nadir of 60% of baseline was reached at approximately 24 hr after initiation of the procedure, consistent with the time course of arousal of comatose patients with fulminant hepatic failure after charcoal hemoperfusion therapy. Analysis of the computer-simulated effects of four different treatment schedules (4 hr/day; 12 hr, then 4 hr/day; 24 hr, then 4 hr/day; and continuous) during a 5-day period showed that continuous hemoperfusion was the most efficient, reducing the plasma unconjugated bilirubin concentration to 1.2 mg% and the extrahepatic, extravascular bilirubin pool to values within the normal range by 24 hr. The usually employed 4-hr regimen did not achieve plasma bilirubin values consistently under 6 mg%, and did not bring the extravascular pool within the normal range until after the fourth treatment. Initial perfusions of either 12 or 24 hr, followed by maintenance treatments of 4 hr/day would achieve normalization of the extrahepatic, extravascular pool within 28 and 24 hr, resp., of initiating treatment. Alternatively, two perfusions of 6 hr each, at the start of treatment and at 12 hr would result in normalization of the extravascular pool by 30 hr. The results suggest that albumin-agarose hemoperfusion could reduce the plasma unconjugated bilirubin concentration substantially and the extrahepatic, extravascular bilirubin pool size to normal in patients with type I Crigler-Najjar syndrome. These results are dependent upon both the efficiency of the device and on the treatment schedule.

8454 HEPATITIS B SURFACE ANTIGEN AND α-FETOPROTEIN IN PATIENTS WITH HEPATOCELLULAR CARCINOMA [Letter to Editor]. (Eng.) Chen, D. S.; Sung, J. L.; Kubo, Y.; Okuda, K. (Coll. Medicine, Natl. Taiwan Univ., Taipei, Taiwan, 100, Republic of China). *Gastroenterology* 74(1):163-165; 1978.

8455 CELL MEDIATED IMMUNITY TO BILE PROTEIN IN THE PATIENTS WITH CHRONIC INTRAHEPATIC CHOLESTASIS, WITH SPECIAL REFERENCE TO PRIMARY BILIARY CIRRHOSIS [Abstract]. (Eng.) Sano, M.; Ito, K. (Sch. Medicine, Kyoto Univ., Kyoto, Japan). *Gastroenterol. Jpn.* 12(4):334; 1977.

8456 VOLATILE METABOLITES IN HEPATIC ENCEPHALOPATHY [Abstract]. (Eng.) Goldberg, E. M.; Wolman, S.; Sandler, S.; Blendis, L. M. (Dept. Medicine, Univ. Toronto, Toronto, Ontario, Canada). *Clin. Res.* 25(5):687A; 1977.

8457 RIGHT HEPATIC LOBECTOMY--7 CASES [Abstract]. (Eng.) Raute, M.; Kersting, K. H.; Trede, M. (Klinikum Mannheim der Universitat Heidelberg, Theodor-Kutzer-Ufer, D-6800 Mannheim, W. Germany). *Langenbecks Arch. Chir.* 395:583; 1977.

8458 POTENTIAL HAZARD OF THERAPY FOR REYE'S SYNDROME WITH UREA CYCLE INTERMEDIATES [Letter to Editor]. (Eng.) Glasgow, A. M. (Children's Hosp. Natl. Medical Center, 2125 13th St. N.W., Washington, DC 20009). *Pediatrics* 60(4):557-558; 1977.

8459 PLASMA AMINO ACID CHANGES AS A FUNCTION OF HEPATOCELLULAR DAMAGE [Abstract]. (Eng.) Milsom, J. P.; Morgan, M. Y.; Sherlock, S. (Royal Free Hosp., Pond St., Hampstead, London NW3 2QG, England). *Clin. Sci. Mol. Med.* 53(3):15P; 1977.

8460 SERUM FERRITIN IN THE DIAGNOSIS OF HEMOCHROMATOSIS [Abstract]. (Eng.) Powell, L. W.; Halliday, J. W.; Russo, A.; Cowlishaw, J. (Dept. Medicine, Univ. Queensland, Brisbane, Australia). *Gastroenterology* 73(5):1239; 1977.

8461 REPTILASE TIME IN CIRRHOSIS AND HEPATOCELLULAR CARCINOMA. (Eng.) Johnson, P. J.; White, Y.; Woolf, I. L.; Williams, R. (King's Coll. Hosp., Medical Sch., London SE5, England). *Br. Med. J.* 2(6091):869-870; 1977.

8462 CONGENITAL HEPATIC FIBROSIS AND CHOLEDOCHUS CYST. (Eng.) Lake, D. N. W.; Smith, P. M.; Wheeler, M. H. (Llandough Hosp., Penarth, Cardiff, Wales). *Br. Med. J.* 2(6097):1259-1260; 1977.

8463 RADIOISOTOPE LIVER IMAGING [Abstract]. (Eng.) McCready, V. R. (Royal Marsden Hosp., Downs Rd., Sutton, Surrey, England). *Br. J. Radiol.* 51(601):64; 1978.

8464 PORTOVENOGRAPHY IN LIVER DISEASES [Abstract]. (Eng.) Ngan, H.; Wong, K. P. (Queen Mary Hosp., Hong Kong). *Br. J. Radiol.* 51(601):65; 1978.

8465 THE VALUE OF HEPATIC ANGIOGRAPHY [Abstract]. (Eng.) Laws, J. W. (King's Coll. Hosp., Denmark Hill, London SE5, England). *Br. J. Radiol.* 51(601):64-65; 1978.

8466 COMPUTED TOMOGRAPHY OF THE LIVER [Abstract]. (Eng.) Fawcitt, R. A. (Dept. Diagnostic Radiology, Univ. Manchester, Manchester, England). *Br. J. Radiol.* 51(601):64; 1978.

8467 ULTRASOUND OF THE LIVER [Abstract]. (Eng.) Berger, L. A. (Royal Free Hosp., London, England). *Br. J. Radiol.* 51(601):64; 1978.

8468 THE USE OF PROFOUND HYPOTHERMIA AND CIRCULATORY ARREST FOR HEPATIC LOBECTOMY IN INFANCY. (Eng.) Ward, C. F.; Arkin, D. B.; Benumof, J. L. (Univ. California Medical Center, 225 W. Dickinson St., San Diego, CA 92103). *Anesthesiology* 47(5):473-474; 1977.

8469 CEREBRO-HEPATO-RENAL SYNDROME: ALSO A DISTAL AXONOPATHY [Abstract]. (Eng.) Partin, J. S.; McAdams, A. J.; Berry, H.; Partin, J. C. (Children's Hosp. Medical Center, Univ. Cincinnati, Cincinnati, OH). *Gastroenterology* 73(5):1238; 1977.

8470 REGENERATION IN THE SIMPLE LIVER ACINUS AFTER SELECTIVE ZONAL INJURY [Abstract]. (Eng.) Nostrant, T. T.; Miller, D. L.; Appelman, H. D.; Gumucio, J. J. (Section Gastroenterology, Univ. Michigan, Ann Arbor, MI). *Gastroentcrology* 73(5):1236; 1977.

8471 BLOOD POLYAMINE DETERMINATIONS IN CHILDREN WITH LIVER DAMAGE [Abstract]. (Eng.) Garnica, A. D.; Rodgers, B. M.; Rennert, O. M. (Univ. Florida Coll. Medicine, Gainesville, FL). *Pediatr. Res.* 12(4, Part 2):434; 1978.

8472 HEPATIC LEFT LOBE LESIONS--CLINICAL USEFULNESS OF MULTIPLE IMAGING TECHNIQUES TO INCREASE LESION DETECTABILITY. (Eng.) Gooneratne, N. S.; Quinn, J. L., III (South Carolina Hosp., 171 Ashley Ave., Charleston, SC 29403). *Clin. Nucl. Med.* 2(11):377-380; 1977.

8473 CONGENITAL CYSTIC DISEASE OF THE LIVER: ISOTOPE SCANNING, ULTRASONOGRAPHY AND ANGIOGRAPHY. (Eng.) McLoughlin, M. J.; Miskin, M.; Thompson, C. P. (Toronto General Hosp., Toronto, Ontario, Canada). *J. Can. Assoc. Radiol.* 28(4):243-248; 1977.

8474 ACCUMULATION OF ^{99m}Tc-DIPHOSPHONATE IN HEPATIC NEOPLASM. (Eng.) Ghaed, N.; Marsden, R. J. (Fitzsimons Army Medical Center, Denver, CO). *Radiology* 126(1):192; 1977.

See also, 7989, 8071, 8140, 8144, 8145, 8170, 8174, 8243, 8251, 8288, 8434, 8484, 8488, 8519, 8520, 8561, 8591, 8599, 8601, 8602, 8603, 8604, 8605.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

8475 PHYSIOLOGIC JAUNDICE OF THE NEWBORN: RELATION TO MATERNAL SERUM AND AMNIOTIC FLUID ALPHA-FETOPROTEIN. (Eng.) Goldstein, A. I.; Farrell, R. C. (Dept. Obstetrics and Gynecology, Univ. California, Irvine, CA 92717). *Obstet. Gynecol.* 51(3):315-317; 1978.

The relationship between physiologic jaundice of the newborn and elevated levels of maternal serum and amniotic fluid alpha-fetoprotein was studied. Eighty-seven paired serum and amniotic fluid specimens taken between 37 and 41 weeks of pregnancy were evaluated. There was an excellent correlation between increased levels of maternal serum and amniotic fluid alpha-fetoprotein and physiologic indirect neonatal hyperbilirubinemia. The maternal serum alpha-fetoprotein levels at 37, 38, 39, 40, and 41 weeks were 384 ± 22, 324 ± 19, 310 ± 23, 272 ± 18, and 253 ± 12 ng/ml, resp., in cases where the neonatal serum bilirubin level was less than 7 mg/100 ml, whereas the values were 451 ± 26, 415 ± 22, 403 ± 16, 387 ± 19, and 344 ± 28 ng/ml in

cases where the neonatal bilirubin level was over 7 ng/100 ml. The amniotic fluid alpha-fetoprotein levels were 394 ± 32, 327 ± 29, 277 ± 26, 271 ± 18, and 258 ± 17 ng/ml in cases in which the bilirubin level was less than 7 ng/100 ml, whereas the levels were 692 ± 16, 681 ± 24, 630 ± 12, 586 ± 14, and 559 ± 17 ng/ml in cases of hyperbilirubinemia. The alpha-fetoprotein assay might be used as an index of fetal liver maturity when considering elective cesarean section or induction.

LIVER AND BILIARY TRACT

Nonviral Hepatitis and Hepatic Injury

8476 ANGIOGRAPHIC ABNORMALITIES AFTER PUNCH BIOPSY OF THE LIVER. (Fre.) Cremniter, D.; Chatel, A.; Bigot, J. M.; Lacombe, P.; Helenon, C. (Hopital Tenon, 4, rue de la Chine, 75970 Paris Cedex 20, France). *J. Radiol. Electrol. Med. Nucl.* 59(1):33-38; 1978.

Vascular abnormalities that developed in nine patients after puncture biopsies of the liver and in one patient after pleural biopsy were detected by angiography. An arterioportal fistula was the abnormality observed in seven patients; the fistula was usually small and disappeared spontaneously. Most often, these fistulas were found accidentally when patients underwent angiography for symptoms related to other pathology. Two patients had hematomas, one of which was associated with an arterioportal fistula. The patient who had a pleural puncture developed a false aneurysm of the distal branch of the hepatic artery and peritoneal hemorrhage. When this patient underwent surgery, a tear in the liver was found that was attributed to the pleural puncture biopsy. Arterioportal fistulas are observed in 60% of patients who undergo angiographic studies within 1 week of liver biopsy. They are rarely serious since they heal rapidly and spontaneously.

8477 DRUG-INDUCED HEPATIC DAMAGE. (Ger.) Keller, J. (Medizinische Universitatsklinik, Hugstetter Strasse 55, 7800 Freiburg, W. Germany). *Therapiewoche* 28(5):762-778, *passim*; 1978.

A review of drug-induced hepatic damage is presented. Drug-induced hepatic damage may be classified according to its clinical features, the histology of the damaged liver tissue, or the mechanism causing the damage. Drugs causing a predictable reaction include cytotoxic drugs with direct hepatotoxicity such as carbon tetrachloride. Drugs causing indirect hepatotoxicity may be cytotoxic or cholestatic, such as tetracycline, methotrexate, *Amanita phalloides*, anabolics, and contraceptives. Drugs that cause unpredictable damage are those that provoke allergic reactions, hyperergy, or metabolic disturbances; some examples are halothane, oxyphenisatin, chlorpromazine, and isonicotinolyhydrazide (INH). Reactions provoked by substances causing predictable damage are characterized by a brief interval between exposure and manifestation of hepatic damage, clear dose-dependence, experimental reproducibility, and nearly universal reaction by exposed persons. Reactions to drugs causing unpredictable damage are generally not dose-dependent and cause allergic hepatic damage or metabolic damage. Diagnostic characteristics of both types are given. A few drugs worthy of special note have been selected for comment; their use and capability for causing liver damage are explained. Among these are halothane, paracetamol, INH, alpha-methyldopa, i.v. tetracycline, oral contraceptives, chlorpromazine, *N*-propyl ajmalin and derivatives, aprinidine, azathioprine, methotrexate, and oxyphenisatin.

8478 A MORPHOLOGIC STUDY OF UNEXPLAINED HEPATITIS FOLLOWING HALOTHANE ANESTHESIA. (Eng.) Wills, E. J.; Walton, B. (Royal Prince Alfred Hosp., Missenden Rd., Camperdown, New South Wales 2050, Australia). *Am. J. Pathol.* 91(1):11-32; 1978.

Liver biopsy specimens from 16 patients who developed hepatitis following halothane anesthesia were examined histologically and ultrastructurally to determine whether the liver morphology could aid in the differential diagnosis between halothane hepatitis and acute viral hepatitis. Ten cases were fatal, although only seven patients died in hepatic failure. Necrosis was not in general disproportionately severe compared with the clinical and biochemical condition of these patients, in contrast with the observations of previous cases of halothane hepatitis. However, two patients with clinically equivocal hepatitis had definite morphologic evidence of hepatic inflammation and necrosis. Necrosis tended to be spotty, but in subjects who died rapidly, confluent necrosis was more clearly delineated. Fatty infiltration was unimpressive. Large numbers of eosinophils were seen in two cases. Cholestasis was too varied in degree and localization to be of diagnostic value. Bile ducts were frequently infiltrated with neutrophils and there was often accompanying erosion of the

parenchymal limiting plate by inflammatory cells. Increased portal tract connective tissue was sometimes as apparent as early as 12 days after the onset of jaundice. Light microscopy of biopsy specimens from three patients who died from unrelated conditions when their liver disease was clinically improving showed a mode of resolution similar to that in viral hepatitis. Bile pigment was seen ultrastructurally in the parenchymal cells with the usual attendant canalicular changes in jaundiced patients. Autophagic activity in hepatocytes and Kupffer cell abnormalities were present in some cases. Dilated rough endoplasmic reticulum was observed in most cases and was the most conspicuous feature in specimens obtained within 14 days of the onset of jaundice. Small defects in the mitochondrial membrane were observed in one patient, and variations in mitochondrial size and shape, abnormalities of their cristae, and crystalline inclusions were striking in only one case. Thus, the results do not support the contention that the mitochondria are the most severely affected organelles in halothane hepatitis. Since lipid vacuolation and a predominantly centrilobular distribution of necrosis are not classically described in fatal viral hepatitis, the presence of these features in some of the present fatal cases was of some diagnostic interest. In general, however, the light and electron microscopic features in patients with unexplained postoperative hepatitis have little diagnostic value.

8479 HALOTHANE AND ANTITUBERCULOUS DRUGS--A HEPATOTOXIC COMBINATION? (Ger.) Schmidt-Wilcke, H. A.; Wolfert, W.; Grundmann, E. (St.-Franziskus-Hospital Munster, Hohenzollernring 72, D-4400 Munster, W. Germany). *Z. Gastroenterol.* 15(8):504-511; 1977.

Two women who were being treated with isoniazid (400 mg/day) and rifampicin (600 mg/day) or ethambutol (1,200 mg/day) for tuberculous arachnoiditis of the optic chiasm and lung tuberculosis, resp., developed toxic liver damage a few days following halothane anesthesia for corneal transplantation and for the treatment of an abscess of a cholecystectomy scar, resp. Both patients had normal liver function before the surgery. Jaundice, pathologically increased serum ammonia, bilirubin, and SGOT levels, and grade III hepatic encephalopathy with somnolence, disorientation, and amnesia were observed. Australia antigen was not found. One patient had undergone halothane anesthesia before tuberculostatic treatment without any complication. Liver biopsy revealed alterations (focal or centrilobular/intermedial liver cell necrosis) compatible with drug hepatitis. The findings did not resemble, however, typical isoniazid hepatitis or typical halothane hepatitis, which suggests the occurrence here of combined toxic effects of halothane and isoniazid on the liver. Complete remission was achieved in both cases in 10-28 days by low-protein diet and lactulose treatment. The absence of literature data concerning an increased incidence of hepatotoxic complications due to the simultaneous use of isoniazid and halothane suggests the possibility of a particular predisposition to this phenomenon in these patients.

8480 LIVER DAMAGE FROM LONG-TERM METHYLTESTOSTERONE [Letter to Editor]. (Eng.) Bird, D. R.; Vowles, K. D. J. (Royal Devon and Exeter Hosp., Exeter, Devon EX2 5DW, England). *Lancet* 2(8034):400-401; 1977.

8481 PORTAL HYPERTENSION WITHOUT LIVER CIRRHOSIS DUE TO CHRONIC VITAMIN A POISONING. (Dut). van den Brandt-Gradel, V. (No affiliation given). *Ned. Tijdschr. Geneeskd.* 122(1):25; 1978.

See also, 8456, 8495.

LIVER AND BILIARY TRACT

Acute Viral Hepatitis

8482 RECENT FINDINGS ABOUT THE EPIDEMIOLOGY OF VIRAL HEPATITIS. (Ger.) Baumgarten, R.; Fengler, J. D. (Stadtisches Krankenhaus Prenzlauer Berg, Nordmarkstrasse 15, DDR-1055 Berlin, E. Germany). *Z. Gesamte Inn. Med.* 32(22):627-630; 1977.

A brief review of the present state of knowledge regarding the infectivity and transmission of viral hepatitis, sterilization and disinfection to protect against it, hepatitis B surface antigen (HBsAg)-carriers, and the risk of acquiring hepatitis in the health professions is presented. Hepatitis A (HA) and hepatitis B (HB) are caused by two different organisms; cross-immunity does not exist. Although both diseases are contracted by both fecal-oral and parenteral routes, the former is predominant in HA, and the latter in HB. Incubation time and the length of the infectious period of both diseases are discussed. HBsAg carriers may be symptom-free. Hepatitis viruses are relatively resistant to heat and chemical agents. The best method of avoiding transmission by way of fomites

is by use of disposable equipment. Physical sterilization methods offer better protection than do chemical methods. The causative agent of HA is generally only excreted briefly by patients except in carriers; however, the HB virus may persist in patients for a long time, serving as a constant source of infection. Persons in the health professions face the greatest risk of contracting viral hepatitis. Patients posing the greatest threat as sources of infection are, in order, chronic dialysis patients, drug addicts, hepatitis patients without demonstrable HBsAg, and HBsAg positive patients. Measures that should be undertaken in dialysis units to reduce the risk of transmission are outlined.

8483 SERO-EPIDEMIOLOGY OF ACUTE VIRAL HEPATITIS. (Ger.) Mueller, R.; Willers, H.; Hoepken, W. (Medizinische Hochschule Hannover, Karl-Wiechert-Allee 9, D-3000 Hannover 61, W. Germany). *Munch. Med. Wochenschr.* 120(15):517-520; 1978.

A sero-epidemiological survey was conducted of 329 patients from the Hannover area, comprising virtually all patients from this area with clinically demonstrated acute viral hepatitis in 1975. Antibodies to hepatitis A virus (anti-HA), cytomegalovirus, and the hepatitis B surface antigen (HBsAg) were determined in all sera; IgM levels were also measured. In HBsAg-negative sera, antibodies to HBsAg (anti-HBs) and to HB core antigen (anti-HBc) were determined also. The diagnosis of hepatitis A (56 cases) was made only in cases in which seroconversion or a fourfold increase in anti-HA titer was observed during the course of the disease; in cases in which only a single serum sample was available, the demonstration of increased IgM levels in addition to the presence of anti-HA sufficed for the diagnosis. Contamination of the population with hepatitis A virus was high and increased with age, despite the low incidence of acute hepatitis A disease. Hepatitis A patients contracted the disease by contact with hepatitis A infections or during trips to southern Europe. All HBsAg-positive cases in this study (211 cases) were recognized as hepatitis B, as neither seroconversion nor significant increases in anti-HA titer were measured during the course of the disease. Non-A, non-B hepatitis was diagnosed in 62 patients. Fifty percent of the hepatitis B patients and 42% of the non-A, non-B hepatitis patients had been exposed parenterally to potentially infectious agents.

8484 VIRAL HEPATITIS AND PREGNANCY. (Fre.) Merger, C.; Chadeyron, P. A.; Levy, J. (Hopital Foch, 40 rue Worth, F 92151, Suresnes, France). *J. Gynecol. Obstet. Biol. Reprod.* [*Paris*] 1(4):529-536; 1977.

The epidemiology, symptoms, and prognosis of mother and infant were studied in nine women who developed long-incubation viral hepatitis during pregnancy. Hepatitis B virus (HBV) infection was suspected in four patients who had been inoculated or had blood withdrawn, and contagious hepatitis possibly due to HBV was suspected in the other five patients, only two of whom were known to have been in contact with a source of contagion. Hepatitis A virus was ruled out as a cause because of the incubation periods shown by these patients. Symptoms appeared in the 5th to 36th week of pregnancy and did not differ from those usually observed in viral hepatitis. The disease lasted 15-30 days in seven of nine patients. Three patients were treated at the onset with corticosteroids and one was treated with steroids after a relapse. Neither chronic hepatitis nor serious jaundice was observed in any of the nine patients. Of the nine women, five delivered premature infants (between 26 and 36 weeks of gestation), and prematurity was the primary determinant of fetal prognosis. An infant born at 29 weeks was stillborn and death may have been due to a small retroplacental hematoma associated with a placenta previa and inhalation of amniotic fluid rather than maternal hepatitis. The other infants were normal at birth and not unusually small for the length of the respective pregnancies. Microscopic examination of the nine placentas did not reveal any sign of viral infection. Neonatal progress of the eight surviving infants was slow; they required an average of 16.25 days to recover their birth weights. Excluding the icterus due to birth at 28 weeks, three infants were jaundiced for many days. Since premature delivery is the most serious complication of viral hepatitis in pregnancy, beta-adrenergic drugs should be used with caution in threatened early labor.

8485 MATERNAL TRANSMISSION OF THE HEPATITIS B VIRUS: FREQUENCY AND CONSEQUENCES. (Fre.) Buffet, C.; Larouze, B. (Hopital Antoine-Beclere, 157 rue de la Porte-de-Trivaux, F 92141 Clamart, France). *Gastroenterol. Clin. Biol.* 1(12):1053-1062; 1977.

The literature on the transmission of hepatitis B virus (HBV) from the mother to the fetus or to the neonate is reviewed with respect to frequency, mode of transmission, and consequences. Studies from occidental countries show that hepatitis B surface antigen (HBsAg), whose presence indicates exposure to the virus, is rare in infants with mothers who are chronic carriers of HBsAg but frequent when the mother had acute hepatitis during the third trimester of pregnancy. Studies from oriental countries show that there is a higher percentage of infants with HBsAg born to mothers who are chronic HBsAg carriers. The presence of HBsAg in the serum of the father had no influence on HBsAg level in the infant. Transmission of HBV *in utero* is suggested by the fact that blood obtained from umbilical cord is positive for HBsAg, the frequency of this event varying from study to study. Some neonates have HBsAg-positive cord blood although their mothers were HBsAg-negative. Electron microscopy has revealed some of the components of Dane particles in cord blood. Amniotic fluid has also been shown to be HBsAg-positive. One study showed an elevated probability of an infant being HBsAg-positive up to 6 months of age if the cord blood was also positive. The possibilities that HBV is transmitted from mother to infant via blood ingested by the infant during birth or via penetration through skin scratches during early life

are considered. Transmission via maternal milk does not appear to occur since HBsAg has not been found in human milk. Maternal transmission of HBV has no immediate serious consequences. Infants born to HBsAg-positive mothers are usually asymptomatic HBsAg carriers with moderate and fluctuating elevations in transaminase levels. Chronic hepatitis (persistent or active) and cirrhosis seem to be rare outcomes; hepatitis usually occurs between 2 and 5 months after birth and is serious, with risk of hepatic coma, chronic hepatitis, and postnecrotic cirrhosis. Malformations such as biliary atresia have been found in infants born to mothers who were HBsAg-positive but statistical studies have not been performed. The risk of primary liver cancer (PLC) is greater when the mother is HBsAg-positive and the father negative for antibody to HBsAg. Families of patients with PLC have elevated levels of HBsAg and antibody to HB core Ag.

8486 IMMUNOLOGICAL ASPECTS AND PROPHYLACTIC AND THERAPEUTIC PROBLEMS OF VIRAL HEPATITIS. (Ita.) Fiaccadori, F.; Pizzaferri, A. (Universita degli Studi di Parma, Parma, Italy). *Minerva Med.* 68(62):4153-4172; 1977.

Problems of viral hepatitis are reviewed and discussed emphasizing hepatitis antigens in serum, the immunological response (humoral and cellular) in hepatitis, and prophylactic and therapeutic implications. Antibody to hepatitis B core antigen (anti-HBc) appears to be an indication of active viral replication whereas antibody to HB surface Ag (anti-HBs) appears to signal a state of induced immunity. The behavior of immunoglobulins in acute and chronic hepatitis, the formation of cytotoxic immune complexes, and the immunological cellular response are analyzed with regard to the relation between changes in the immune system and the clinical course of the disease. It was found that, generally, IgA and IgG levels are within normal limits, while IgM levels are within the normal limits for patients who are HB surface Ag (HBsAg)-positive and are significantly elevated for those who are HBsAg-negative. Standard gammaglobulins are fairly effective against type A hepatitis, and notable protection has been demonstrated in chimpanzees by a vaccine against type B. Standard gammaglobulins are ineffective in HBsAg-positive hepatitis, but gammaglobulins from individuals with high anti-HBs levels in the blood are effective in such cases and have been used successfully to treat fulminant hepatitis.

8487 HUMORAL AND CELL-MEDIATED IMMUNITY TO HEPATITIS B VIRUS ANTIGENS IN ACUTE AND CHRONIC LIVER DISEASE. (Eng.) Aldershvile, J.; Dietrichson, O.; Hardt, F.; Nielsen, J. O.; Skinhoj, P. (Kommunehospitalet, DK-1399 Copenhagen K, Denmark). *Scand. J. Gastroenterol.* 12(8):917-922; 1977.

The specific humoral immune response to hepatitis B virus (HBV) antigens (hepatitis B surface antigen [HBsAg], hepatitis B core antigen [HBcAg], and hepatitis B e antigen [HBeAg]) was correlated with the cellular immunity to HBsAg and the clinical course of the HBV infection. Twelve patients were investigated after clinical and biochemical recovery from acute viral hepatitis type B and 13 were examined during the acute phase of type B hepatitis; 13 other patients had chronic active hepatitis with or without cirrhosis, 8 patients had chronic active non-B liver disease, 10 patients had miscellaneous HBV-negative liver diseases, and 12 subjects were healthy volunteers. None of the 13 patients examined during the acute phase of type B hepatitis had antibody to HBsAg (anti-HBs), but all 12 convalescent patients had anti-HBs. All patients had anti-HBc at all phases of viral hepatitis. HBeAg was present transiently in one patient. A cellular immune response to HBsAg (migration index <0.93) was found in 4 of 13 patients during the acute phase and in 7 of the 12 patients in the convalescent phase. Seven patients were studied longitudinally for cellular immune response to HBsAg. During the acute phase, migration inhibition was found in two patients only. In contrast, at the time the HBsAg was cleared in six patients, five had a cellular immune response to HBsAg. One of the nonresponding patients developed persistent hepatitis B surface antigenemia, and the other patient had been HBeAg-positive for several weeks. After HBsAg clearance, the migration index returned to normal in all five patients with previous inhibition. Only one patient with chronic type B hepatitis showed a cellular immune response to HBsAg, which was similar to the findings in two patients in each of the two remaining groups. The latter results suggest that a cellular immunity to HBsAg is not a prerequisite for the development of these conditions.

8488 IMMUNOGLOBULINS IN SUBMASSIVE HEPATIC NECROSIS. (Eng.) Kaymakcalan, H.; Dourdourekas, D.; Szanto, P. B.; Ainis, H.; Steigmann, F. (Cook County Hosp., 1825 W. Harrison St., Chicago, IL 60612). *Am. J. Gastroenterol.* 69(3, Part 1):289-294; 1978.

Immunoglobulin levels were determined in 34 patients with acute viral hepatitis and correlated with other parameters such as the levels of serum total globulins, gamma globulins, hepatitis B surface antigen (HBsAg), and enzymes to determine whether the immunoglobulin levels could be of prognostic or diagnostic significance. Seventy-three percent of the 15 patients with typical acute viral hepatitis (as determined by liver biopsy) had a slightly elevated serum globulin level (mean 3.6 g/100 ml). IgG was elevated above normal in 13 of the 15, ranging between 1,325 and 2,450 mg/100 ml in these patients, and was normal in the other two (1,010 and 1,210 mg/100 ml, resp.). The IgM level was elevated in 14 patients, the mean level being 305 mg/100 ml in all these patients. Of the 19 patients with submassive hepatic necrosis, 47% showed low serum globulin levels (mean 3.21 g/100 ml). The IgG levels were below the normal range in seven patients, within normal limits in six, and slightly elevated in six. Among the latter six patients, three showed only

very slight elevations (1,300, 1,360, and 1,385 mg/100 ml, resp.), whereas the other patients had levels of 1,475, 1,800, and 1,650 mg/100 ml, resp. The IgM levels were moderately elevated in 14 patients (mean 279 mg/100 ml). The difference between the IgG levels in the two groups was statistically significant ($p<0.01$). Only 31.55% of the 19 patients with submassive liver necrosis showed slight elevations of the IgG level, while 86% of patients with classical viral hepatitis had elevated levels. These results suggest that the differential diagnosis of submassive hepatic necrosis in viral hepatitis from classical viral hepatitis can be aided by determining the serum IgG level.

8489 ACUTE AND CHRONIC HEPATITIS REVISITED: REVIEW BY AN INTERNATIONAL GROUP. (Eng.) Scheuer, P. J.; Bianchi, L.; De Groote, J.; Desmet, V. J.; Gedigk, P.; Korb, G.; *et al.* (Royal Free Hosp., London NW3 2QG, England). *Lancet* 2(8044): 914-919; 1977.

The pathological features of chronic hepatitis and their importance in the development of cirrhosis are reviewed. The development of chronic liver disease following acute hepatitis is also discussed. Chronic persistent hepatitis is a term referring to portal inflammation, with preserved lobular architecture and little or no fibrosis. There is little likelihood of direct progression to cirrhosis. A new category, chronic lobular hepatitis (CLH) had been proposed to describe the liver lesion in patients with prolonged hepatocellular dysfunction in whom the pathological changes are mainly confined to the lobules. These changes comprise spotty necrosis and inflammation as found in acute viral hepatitis. Piecemeal necrosis and bridging necrosis are not regarded to be part of the CLH lesion. Chronic aggressive hepatitis (CAH) has been redefined as a chronic inflammatory and fibrosing liver lesion of varied etiology and variable histological features. The features common to all untreated examples are piecemeal necrosis together with new fiber formation and lymphocytic infiltration of portal tracts and lobules. Other infiltrating cells may also be found, and features of acute hepatitis may be superimposed. Passive septa formed by collapse after bridging or multilobular liver cell necrosis may be present. Cirrhosis is not a defining criterion but may develop. Signs of liver cell regeneration are usually seen in CAH, and the borderline between CAH and cirrhosis in the more severe cases is not sharp. The confluent type of necrosis (i.e., central-central bridging, central-portal bridging, or multilobular necrosis) seen in acute hepatitis is often followed by healing and sometimes by complete recovery. In piecemeal necrosis however, where there are portal-to-portal connections, the development of chronic liver disease is likely. The authors believe that the morphological feature of greatest importance in the evolution of chronic hepatocellular disease is the infiltration of the tissue by lymphocytes and plasma cells that accompanies piecemeal necrosis and probably represents a continuing immunological reaction, at least in viral cases. The process by which acute hepatitis evolves into chronic liver disease requires careful analysis in different centers, so that the natural history and response to treatment of the different components can be accurately determined.

8490 THE CLINICAL EVOLUTION OF VARIOUS FORMS OF HEPATITIS. (Ger.) Maier, K. P. (Medizinische Universitatsklinik, Hugstetter Strasse 55, 7800 Freiburg, W. Germany). *Therapiewoche* 28(5): 738-746, *passim*; 1978.

Two forms of acute hepatitis, hepatitis A (HA) and hepatitis B (HB), may be distinguished from each other on the basis of the presence of HB surface antigen (HBsAg) in the blood of HB patients and of HA antigen (HAAg) in the blood of HA patients. A third form, non-A, non-B hepatitis may also be distinguished and may be more than one entity; it appears mostly as post-transfusion hepatitis. In HB patients, antibodies to the HB virus (HBV) core antigen (anti-HBc) may be found in the serum even after HBsAg has disappeared. The antibody against HBsAg (anti-HBs), may appear months after an acute HB attack. In a patient with suspected acute hepatitis, the transaminase levels are determined first. The Australia antigen test for determination of HBsAg follows demonstration of high transaminase levels. If both tests are positive, the patient is classified as having acute HB. Ninety percent of acute hepatitis patients recover spontaneously; medicinal therapy does not exist for this disease. Some patients evolve chronic hepatitis; HB patients are more at risk than are hepatitis A patients in this respect. In cases of chronic hepatitis, the presence of HA or HB must be determined. If the disease is neither HB nor HA, it may be chronic aggressive hepatitis (CAH) or chronic persistent hepatitis; the former must be treated, while the latter generally heals itself. Therapy for CAH with transaminase levels more than 10 times the normal value, SGPT 5 times the normal value, and an increase in gammaglobulin levels consists of administration of prednisolone by itself or in combination with azathioprin generally for more than 1 yr.

8491 NON-A, NON-B HEPATITIS. (Eng.) Feinstone, S. M.; Purcell, R. H. (Natl. Inst. Allergy Infectious Disease, Natl. Inst. Health, Bethesda, MD 20014). *Ann. Rev. Med.* 29:359-366; 1978.

Evidence that at least one additional agent, antigenically unrelated to hepatitis A or B viruses, is etiologically responsible for human viral hepatitis is reviewed. The epidemiology of this non-A, non-B hepatitis resembles that of type B hepatitis more than that of type A disease. Non-A, non-B hepatitis frequently follows blood transfusion and can progress to a chronic state, which makes its control highly important. Attempts to identify an antigen specific for non-A, non-B hepatitis or to transmit the disease to nonhuman primates have so far been unsuccessful. The only known means to prevent this disease is by the exclusive use of volunteer sources of blood for

transfusions. The efficacy of immune serum globulin in preventing non-A, non-B post-transfusion hepatitis is poorly defined. Other nonspecific methods for screening blood donors, such as measuring enzyme levels, are currently under investigation.

8492 IDENTIFICATION OF HEPATITIS A VIRUS IN FECES: DIAGNOSTIC AND EPIDEMIOLOGICAL VALUE. (Fre.) Coursaget, P.; Maupas, P.; Hibon, P.; Goudeau, A.; Sizaret, P. Y.; Despert, F. (Hopital Pediatrique Gatien de Clocheville, F 37000 Tours, France). *Nouv. Presse Med.* 6(44):4109-4113; 1977.

The presence of virions was detected by immunoelectron microscopy in the feces of 11/13 patients with hepatitis A. Fecal samples were suspended in a hydrolysate of lactalbumin, and the viral-containing fraction was concentrated by successive centrifugations. Polyethyleneglycol 6000 was added to the viral suspension to a final concentration of 10%. The viral particles were observed ultrastructurally after adding anti-hepatitis A virus immune serum (prepared from a patient with hepatitis A 2 months after onset of icterus), incubation at 37 C for 1 hr, concentration by centrifugation, and staining with uranyl acetate. The two negative fecal samples were collected 1 month after onset of clinical signs of hepatitis. Samples with highest concentrations of virus particles were collected during the preicteric phase of the disease. The limit of the period of detection of virus was 5 days after onset of jaundice. There appeared to be a correlation between elevation of SGPT level and the presence of virions. A kinetic study of the appearance of antibodies was performed in one patient. Specific antibodies against hepatitis A virus reached a maximum 2 months from the time of onset of jaundice.

8493 e ANTIGEN (e)/e ANTIBODY (ANTI-e) AND TRANSMISSION OF HEPATITIS B AMONG HOUSEHOLD CONTACTS [Abstract]. (Eng.) Perrillo, R.; Ellis, F.; Clay, P.; Wellinghoff, W.; Gelb, L.; Aach, R. (Cochran Veterans Admin. Hosp., St. Louis, MO). *Gastroenterology* 73(5):1239; 1977.

8494 CARCINOEMBRYONIC ANTIGEN (CEA): A SENSITIVE TEST FOR HEPATITIS [Abstract]. (Eng.) Molnar, I. G.; Gitnick, G. L. (Univ. California, Los Angeles, CA). *Gastroenterology* 73(5):1235; 1977.

8495 ETIOLOGY OF ACUTE HEPATIC FAILURE. (Eng.) Depew, M. C. (Acute Hepatic Failure Study Group, Univ. Southern California, Los Angeles, CA). *Gastroenterology* 73(5):1236; 1977.

8496 RELATIONSHIP BETWEEN SERUM α-FETOPROTEIN AND e ANTIGEN IN ACUTE AND CHRONIC HEPATITIS B INFECTION [Abstract]. (Eng.) Miller, D. J.; Bloomer, J. R.; Williams, A. E.; Waldmann, T. A.; McIntire, K. R.; Klatskin, G. (Dept. Medicine, Yale Univ., New Haven, CT). *Gastroenterology* 73(5):1234; 1977.

8497 VIRAL HEPATITIS, TYPE B CARRIER STATE--A PSYCHOSOCIAL PROBLEM [Abstract]. (Eng.) Powers, R.; Kiernan, T. (Veterans Admin. Hosp., East Orange, NJ). *Gastroenterology* 73(5):1240; 1977.

8498 IMMUNOGLOBULINS AND COMPLEMENT LEVELS IN ACUTE VIRAL HEPATITIS [Abstract]. (Eng.) Meyer, O.; Keynan, A.; Skibin, A.; Chattah, F.; Quastel, M. R. (Soroka Medical Center, Beersheba, Israel). *Isr. J. Med. Sci.* 13(10):1044; 1977.

8499 ANTIVIRAL THERAPY IN HEPATITIS B INFECTION [Abstract]. (Eng.) Merigan, T. C.; Smith, J. L.; Pollard, R. B.; Neal, E. A.; Sawhney, V.; Gregory, P. B.; Robinson, W. S. (Stanford Univ. Medical Center, Stanford, CA 94305). *J. Supramol. Struct.* (Suppl. 2):229; 1978.

8500 INHIBITION OF HEPATITIS B DNA POLYMERASE BY INTERCALATING AGENTS. (Eng.) Hirschman, S. Z.; Garfinkel, E. (Mount Sinai Sch. Medicine, New York, NY 10029). *Nature* 271(5646):681-683; 1978.

8501 HEPATITIS B ANTIGEN AND ANTIBODY: PREVALENCE AMONG NEW YORK DENTISTS. (Eng.) Jackson, R. J.; Lyman, D. O.; Weil, R. B.; Pert, J. H.; Stevens, R. W. (Epidemic Intelligence Service, State of New York Dept. Health, Tower Building, Empire State Plaza, Albany, NY 12237). *N.Y. State J. Med.* 78(2):222-224; 1978.

8502 THE SIGNIFICANCE OF ELECTROENCEPHALOGRAPHY IN EVALUATION OF SEVERITY AND COURSE OF VIRAL HEPATITIS. (Rus.) Farber, N. A.; Gubsky, L. V. (D. I. Ivanov Inst. Virology, Acad. Medical Science USSR, Moscow, USSR). *Vestn. Akad. Med. Nauk. SSSR* (9):43-45; 1977.

8503 CHRONIC HEPATITIS AS A CONSEQUENCE OF ACUTE VIRAL HEPATITIS. (Pol.) Zielinska, W. (Kliniki Chorob Zakaznych, Instytutu Chorob Wewnetrznych Am w Gdansku, Gdansk, Poland). *Pol. Tyg. Lek.* 32(37):1431-1433; 1977.

See also, 8144, 8439, 8441, 8442, 8443, 8446, 8454, 8471, 8597.

8504 TREATMENT OF CHRONIC ACTIVE HEPATITIS: AN ANALYSIS OF THREE CONTROLLED TRIALS. (Eng.) Wright, E. C.; Seeff, L. B.; Berk, P. D.; Jones, E. A.; Plotz, P. H. (Veterans Admin. Hosp., 50 Irving St., N.W., Washington, DC 20422). *Gastroenterology* 73(6):1422-1430; 1977.

Three controlled trials of the use of corticosteroids and cytotoxic drugs in the therapy of chronic active hepatitis (CAH) were analyzed as to whether the experiments are designed and executed according to current standards for a controlled study and whether the results can be applied to the entire spectrum of CAB. A trial of no therapy (27 patients) versus prednisolone (PS, 22) at the Royal Free Hospital (RFH) during 1963-1967, a trial of azothioprine (A, 25) versus prednisone (P, 22) at the King's College Hospital (KCH) during 1968-1971, and a trial at the Mayo Clinic (MC) that initially (1967-1970) involved placebo (17) versus P (18) versus A (14) versus P + A (14) and later (1967-1974) added the modality of titrated P on alternate days (PT, 31) with 16, 30, 13, and 30 patients in the other 4 groups, resp., are reviewed in detail. In the RFH trial, the mortality was 56% in the untreated group and 14% in the PS group ($p<0.001$); in the KCH trial, mortality was 5% and 28%, resp., in the P and A groups; in the MC trial, the mortality in the P and P + A groups was significantly lower ($p<0.05$) than in the placebo and A groups, and P + A was shown to be superior to PT in inducing histological remission while causing fewer adverse side-effects. When 16 separate factors of design and execution were assessed, the RFH trial met 5 and partially met 2, the KCH trial met 6 and partially met 3, and the MC trial met 12 of the criteria. Although some of the omissions were unavoidable and the perfect trial probably cannot be executed, the deficiencies must nevertheless affect the results, particularly the lack of a placebo-treated group in the RFH and KCH trials, which in essence tested only drug efficacy, whereas the MC trial also permitted an analysis of risk factors. Summarizing the results of these three trials, the authors conclude that the only CAH patients in whom the benefit from corticosteroid therapy has been shown to outweigh the potential drug toxicity are those who are symptomatic, hepatitis B surface antigen (HBsAg)-negative, and who have severe histological abnormalities (subacute hepatitis with multilobular necrosis or cirrhosis). The authors believe that the results of these studies cannot be applied to the entire spectrum of CAH as currently recognized; biochemical and serological screening procedures are now able to detect CAH among asymptomatic individuals, so that a further evaluation of corticosteroid therapy is required for patients who are asymptomatic, HBsAg-positive, or who have the milder histological lesion of CAH alone.

8505 D-PENICILLAMINE IN THE TREATMENT OF CHRONIC ACTIVE LIVER DISORDERS. (Ita.) Manzillo, G.; Sagnelli, E.; Balestrieri, G. G.; Maio, G.; Izzo, C. M.; Romano, F.; *et al.* (Clinica delle Malattie Infettive dell'Universita di Napoli, Napoli, Italy). *Clin. Ter.* 82(2):117-126; 1977.

The effectiveness of D-penicillamine therapy in patients with chronic active liver diseases (CALD) was studied in 36 patients of whom 27 were hepatitis B surface antigen (HBsAg)-positive; 25/36 were treated with D-penicillamine for 6-12 months and 11/36 terminated the treatment before 6 months. A control group of 17 CALD patients was given placebo for 6-12 months; 15/17 were HBsAg positive. The study group was given D-penicillamine: 300 mg/day for 1 week, 600 mg/day for 1 week, 1,200 mg/day for 1 week, 1,800 mg/day for 4 weeks, and then maintained with 700-900 mg/day. They also received 300 mg/day of vitamin B_6 p.o. throughout the period of treatment. Patients treated with D-penicillamine, whether for 6 or 12 months, had a statistically significant reduction in levels of SGOT and SGPT ($p<0.01$ and $p<0.05$, resp.) Also the serum IgM levels were significantly lower in these patients than in the control group ($p<0.01$). Of the 25 treated for >6 months, histological activity completely disappeared in 7, was reduced in 4, did not change in 13, and increased in 1. In the control group, 4 had a reduction, 11 showed no change, and 2 had an increase in histological activity. Side-effects of the drug were skin eruptions, temporary granulocytopenia in the initial phase of treatment, and proteinuria with or without hematuria. The authors will consider D-penicillamine a valid therapeutic alternative to corticosteroids once their findings are confirmed in a larger series.

8506 SOME MORPHOLOGICAL DATA ON CHRONIC HEPATITIS. (Ita.) Giarelli, L.; Bianchi, C.; Antonutto, G. (Istituto di Anatomia e Istologia Patologica dell'Universita, Trieste, Italy). *Minerva Med.* 68(55):3779-3784; 1977.

Morphological aspects of chronic hepatitis are discussed to contribute a clearer understanding to the term chronic hepatitis. A direct immunofluorescence technique using antibody to hepatitis B surface antigen HBsAg conjugated to fluorescein can detect HBsAg in biopsied liver tissue. In the case of granulomatous hepatitis, an etiological diagnosis is often not possible solely on the basis of morphology. In these cases, tuberculosis is often the cause. Important differences in evaluation tend to be made on biopsy and autopsy materials. With biopsies, there is a tendency to overevaluate the observed modifications, while there is a tendency to underevaluate abnormalities seen at autopsy. Problems are encountered when distinguishing between chronic hepatitis and myelolymphoproliferation. An 82-yr-old man was treated for chronic lymphatic leukemia with antimitotic and cortisone drugs. At autopsy, a generalized cryp-

tococcoidosis was discovered; accumulations of crypto cocci were visible in the intralobular regions of the liver. A biopsy of a 69-yr-old woman, for whom clinical and laboratory data strongly suggested eosinophilic leukemia, revealed large numbers of eosinophils along with other types of cells infiltrating the liver. The liver of a 77-yr-old woman with Waldenstrom's disease showed fibrosis and a lymphoid infiltrate in the portal spaces. At autopsy, a 54-yr-old woman who had suffered for a number of years with cryoglobulinemia had fibrotic portal spaces and a lymphoid infiltrate; the infiltrate was immunofluorescent IgM-positive. These data illustrate that there is a morphological continuum between certain forms of chronic hepatitis and hemolymphoproliferative disease, which makes it difficult to distinguish between these types of lesions. Morphological data can help determine the causes of these disease states.

8507 SOME PECULIARITIES OF THE CONDITION OF THE GASTRODUODENAL REGION IN PATIENTS WITH CHRONIC HEPATITIS (DATA ON ENDOSCOPY AND INTRAGASTRIC pH-METRY). (Rus.) Grigoriev, P. Yu.; Georgievsky, N. I.; Triiger, I. I.; Ledoschuk, B. A.; Chertov, A. D. (Dept. Therapy, Blagoveshchensk Medical Inst., Blagoveshchensk, USSR). *Ter. Arkh.* 48(9):57-61; 1976.

8508 SIGNIFICANCE OF PROTEOLYTIC FACTOR IN THE ORIGIN OF DUODENITIS AND JEJUNITIS IN CHRONIC HEPATITIS AND LIVER CIRRHOSIS. (Rus.) Yakhontova, O. I. (Dept. Hosp. Therapy, Leningrad Inst. Medicine and Hygiene, Leningrad, USSR). *Ter. Arkh.* 48(9):54-57; 1976.

See also, 8071, 8144, 8387, 8439, 8441, 8443, 8446, 8459, 8461, 8466, 8471, 8487, 8489, 8496, 8503, 8518, 8526, 8528.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

8509 DRINKING AND DIETARY HABITS AND PREVALENCE OF SOMATIC ABNORMALITIES IN CHRONIC ALCOHOLICS HOSPITALIZED FOR DETOXIFICATION. (Eng.) Wallerstedt, S. (Sahlgrenska sjukhuset, Univ. Goteborg, S-41345 Goteborg, Sweden). *Acta Hepatogastroenterol.* 25(1):6-12; 1978.

The alcoholic histories, drinking and dietary habits, and physical status of 61 male chronic alcoholics were studied within 3 days after the end of the last debauch. The mean duration of alcoholism was 11 yr. The duration of the debauches usually varied between 1.5 and 3.5 weeks. Forty patients were "intermittent drinkers," who did not admit any alcohol consumption between debauches. None of the patients had chronic liver damage, but steatosis was present in 23 of the 33 patients in whom a liver biopsy was carried out. Only one patient each was found to have fibrosis and cirrhosis, resp. The amount of steatosis was not correlated to any parameter concerning drinking or dietary habits. There were statistically significant correlations between the final daily amount of alcohol consumed and heart rate ($p<0.05$), between the duration of the last debauch and the systolic blood pressure ($p<0.05$) and the diastolic pressure ($p<0.01$), and between the presence of Dupuytren's contracture and the age of the alcoholic ($p<0.01$).

8510 CLINICAL ASPECTS OF ALCOHOLIC HEPATITIS: A REVIEW OF 26 CASES. (Eng.) Demeulenaere, L.; Van Waes, L. (Sint-Andrieskliniek, Krommewalstraat 9, B-8880 Tielt, Belgium). *Acta Gastroenterol. Belg.* 40(7/8):261-270; 1977.

The clinical features in 26 patients with biopsy-proven alcoholic hepatitis are reviewed. Only seven patients were hospitalized because of prominent liver-related symptoms (jaundice, ascites, or abnormal liver function tests). The onset of disease was mostly insidious, and weakness, anorexia, and weight loss were the most common complaints (experienced by 24, 22, and 16 patients, resp.). Right upper quadrant pain was present in 11 patients. The association of fever, abdominal pain, and jaundice in five patients made the differential diagnosis between hepatitis and a surgical biliary condition difficult. Six patients were seen in delirium tremens, and four presented with manifestations of hepatic encephalopathy. Three patients progressed to liver pre-coma or coma. Two patients each developed minor gastric bleeding associated with gastritis or diffuse s.c. hematomas secondary to clotting abnormalities. Hepatomegaly occurred in 16 patients, and 8 were found to have ascites. In five patients, ascites developed in the absence of cirrhosis; all had pericentral venous sclerosis on liver biopsy, and two

also had a low serum albumin level. In six patients, spider angiomata occurred during the acute phase of the disease. SGOT and SGPT levels were elevated above 45 U in 81% and 65% of the cases, resp. Transaminase elevation was usually moderate, and rarely exceeded 10 times the normal upper limit. The gamma-glutamyltranspeptidase level was elevated in 93%. Alkaline phosphatase and bilirubin levels were elevated in 73% and 39%, resp. Two patients (one with cirrhosis) died during the acute phase. Both had marked jaundice, abdominal pain, tender hepatomegaly, and spiking fever on admission. Three of 10 patients with hepatitis and fatty liver who continued drinking progressed to cirrhosis and 1/10 to fibrosis after 3, 6, 8, and 48 months, resp. Four patients who stopped drinking had full clinical and biochemical recovery. Repeat biopsy in two patients who did not abstain showed persistent necrosis and inflammation, but a biopsy in one patient who abstained showed a normal liver. It is concluded that clinical symptoms and biochemical alterations are unreliable indicators of the degree of liver injury in alcoholic hepatitis.

8511 CLINICO-PATHOLOGICAL EXPERIENCE WITH ALCOHOLIC HEPATITIS. (Eng.) McDonald, G. S. A.; Hourihane, D. O. (Div. Lab. Medicine, Trinity Coll., Dublin, Ireland). *Ir. Med. J.* 71(2):47-51; 1978.

A clinico-pathological study was carried out in 32 Irish patients, and differences between these patients and those reported in the American literature are discussed. The most common clinical findings in the 20 Irish patients in whom a liver biopsy was carried out were raised levels of serum enzymes (89%) and a palpable liver (74%). The most common findings reported in a series in the American literature were palpable liver (81%) and pyrexia (56%); the incidence in the American series of nausea and vomiting (55%), abdominal pain (46%), and leukocytosis (56%) was greater than in the Irish patients (35%, 26%, and 19%, resp.). Among the Irish patients, alcoholic hepatitis was diagnosed before the liver biopsy in only one instance. Among the 12 Irish patients in whom alcoholic hepatitis was discovered at autopsy, liver failure due to alcoholic hepatitis with or without liver cirrhosis was the cause of death in 6. Alcoholic hepatitis was recorded as a clinical diagnosis in only 1 of the 12 patients. The pathological diagnosis of alcoholic hepatitis was based on the presence of necrosis and polymorphonuclear infiltration of the parenchyma and portal tracts. Two patients with severe necrosis had central vein phlebitis. Mild to moderate fatty change was present in 30 cases, and severe fatty change occurred in the remaining 2 patients. In contrast, fatty change was present in only 87% of the American cases. Alcoholic hyaline was observed in 45% of the 20 patients who underwent biopsy and in 58% of the autopsy cases; the presence of alcoholic hyaline occurred with a similar frequency in the American series. Fibrosis was present in 85% of the liver biopsy specimens and in 63.9% of the American cases. It appears that the clinical symptoms of alcoholic hepatitis are somewhat less severe in Irish patients in comparison with those in American patients. Such a diagnosis should be considered in alcoholic patients with an enlarged tender liver and raised serum enzyme levels.

8512 ALCOHOLIC HEPATITIS: RECENT PROGRESS IN THE STUDY OF THE PHYSIOPATHOLOGY AND CLINICAL FEATURES OF LIVER DAMAGE INDUCED BY ALCOHOL. (Ita.) Lovisetto, P.; Actis, G. C.; Ferraris, R.; Marchi, L.; Biarese, V. (Istituto di Medicina Interna dell'Universita di Torino, Torino, Italy). *Minerva Med.* 68(62):4127-4146; 1977.

Recent progress in the study of the physiopathology and clinical features of liver damage induced by alcohol is reviewed and discussed with respect to the metabolism of alcohol, the effects of alcohol on the liver, histological characteristics and histogenesis of acute alcoholic hepatitis, and clinical characteristics, laboratory reports, diagnosis, prognosis, and therapy of alcoholic hepatopathy. The clinical picture is usually marked by the sudden onset of nausea, icterus, painful hepatomegaly, fever, ascites, and signs of encephalopathy. Laboratory tests can reveal hyperbilirubinemia, elevated alkaline phosphatase levels, elevated gamma-glutamyl transpeptidase activity, and augmented levels of bile salts. Leukocytosis and hyperuricemia may also be present. Needle biopsy can reveal various degrees of qualitative and quantitative damage (vacuolar degeneration, steatosis, cell necrosis, inflammatory infiltration, and interlaminar hyaline sclerosis) that depends on the extent of alcohol intake. Mallory bodies associated with polymorphonuclear cell infiltration and centrilobular sclerosis are characteristic indications of alcoholic hepatitis. Steatosis is also a common and diagnostic result of chronic alcohol intoxication. Progress of the disease is dependent to a large degree on immunological considerations. Treatment consists of elimination of alcohol, rest, and dietary management (high-protein and high-caloric diet).

8513 BIOPSY-DEMONSTRATED HEPATIC CHANGES IN CHRONIC ALCOHOLISM. (Ger.) Lorenz, G. (Pathologische Institut, Ernst-Moritz-Arndt-Universitat, Fr.-Loeffler-Strasse 23e, DDR-22 Greifswald, E. Germany). *Z. Gesamte Inn. Med.* 33(5):145-148; 1978.

To classify hepatic damage due to alcoholism, random adult liver biopsies obtained during a 5-yr period from 2,952 patients, 506 of whom were clinically demonstrated alcoholics, were examined histologically. Of the alcoholics, 473 were men. The histological findings permitted the grouping of the biopsies into four categories. The first category (showing liver changes with no specific outstanding disease findings) comprised 39% of the biopsies from alcoholic patients; the second (consisting of fatty livers) comprised 40%; the third (alcoholic hepatitis) encompassed 18% of the biopsies; and the fourth (demonstrating cir-

rhosis) comprised 3%. The mean age of persons with alcoholic hepatitis was 40 yr in men, 45 in women. Iron deposits, which are supposed to have great diagnostic value in determining alcoholic hepatic damage, were present in 48% of the cases, and cholestasis was present in 20%. Alcoholic hepatitis developed in most cases in 10-12 yr after the beginning of ethanol abuse. Fatty liver originated predominantly in the abuse of alcohol.

8514 ELECTRON MICROSCOPIC STUDY OF ALCOHOLIC LIVER DISEASE WITH SPECIAL ATTENTION TO THE CHANGES OF MESENCHYMAL CELLS OF THE LIVER. (Eng.) Balazs, M.; Varkonyi, S.; Pinter, A. (Janos Hosp., Diosarok u. 1, H-1125 Budapest, Hungary). *Exp. Pathol. (Jena)* 14(6):340-350; 1977.

Liver biopsy material from 48 patients with chronic alcoholism was studied ultrastructurally by electron microscopy to observe how liver damage is influenced by the disease progression. The patients were grouped on the basis of clinical and histological criteria: group I, 16 patients and alcoholic fatty liver; II, 11 patients, fatty liver with increased mesenchymal activity; III, 6 patients, acute alcoholic hepatitis; IV, 8 patients, chronic alcoholic hepatitis; and V, 7 patients, alcoholic cirrhosis. Fat accumulation, mitochondrial alteration (swelling of mitochondria and elongation and aggregation of cristae), and focal cytoplasmic degradation were demonstrated in all cases with similar frequency and severity and were not correlated with the clinical severity of the disease. However, proliferation of mesenchymal cells, signs of their secretory activity, and fiber formation did correlate with the progression of the disease. In group I, an increase of perisinusoidal fat-storing cells was noted in many cases. In group II, the number of mesenchymal cells was increased; cytoplasm was abundant and rich in organelles. In group III, leukocytes were present in the sinusoidal wall and a basement membrane developed along Kupffer cells without significant development of collagen fibers in the Disse space. The most prominent feature in group IV was the enlargement and proliferation of Kupffer cells; the cytoplasm was very rich in organelles and collagen bundles were seen around the Kupffer cells. In group V, proliferation of Kupffer cells was prominent, and many of the perisinusoidal cells were similar to fibroblasts and protruded deeply between the liver cells; broad collagen bundles were situated in the intercellular space of liver cells. This study suggests that between alcoholic hepatitis and alcoholic cirrhosis there is another stage, the chronic alcoholic hepatitis stage (closely resembling chronic aggressive hepatitis), whose main characteristic is not the severity of the injury of the liver cells but the great proliferation of mesenchymal cells and lymphoid infiltration. There may be prognostic value in identifying those alcoholics whose fatty liver is combined with increased mesenchymal cell activity at the beginning of liver disease, possibly marking the onset of chronic liver disease.

8515 ASSOCIATION BETWEEN HLA-BW40 AND ALCOHOLIC LIVER DISEASE WITH CIRRHOSIS. (Eng.) Bell, H.; Nordhagen, R. (Oslo City Hosp., Oslo 1, Norway). *Br. Med. J.* 1(6116):822; 1978.

8516 THE EFFECT OF PORTACAVAL SHUNTING IN THE ALCOHOLIC CIRRHOTIC ON SERUM CHOLESTEROL CONCENTRATION: A RETROSPECTIVE STUDY. (Eng.) Kuo, J. Y.; Dreiling, D. A. (Mount Sinai Sch. Medicine, City Univ. New York, 100th St. & Fifth Ave., New York, NY 10029). *Am. J. Gastroenterol.* 68(3):270-272; 1977.

8517 THE FATTY LIVER: ROENTGENOLOGICAL RECOGNITION. (Eng.) Anonymous. (No affiliation given). *Am. J. Dis. Child.* 132(1):72; 1978.

8518 IN VIVO AND IN VITRO FIXATION OF IgA ON THE HEPATOCELLULAR MEMBRANE [Abstract]. (Eng.) Hopf, U.; Hutteroth, T. H.; Meyer zum Buschenfelde, K. H. (Freie Universitat Berlin, Klinikum Charlottenburg, Berlin, W. Germany). *Digestion* 16(4):357; 1977.

See also, 8251, 8441, 8442, 8443, 8459, 8461, 8487, 8595.

LIVER AND BILIARY TRACT

Cirrhosis

8519 THE MORPHOLOGY OF CIRRHOSIS: RECOMMENDATIONS OF DEFINITION, NOMENCLATURE, AND CLASSIFICATION BY A WORKING GROUP SPONSORED BY THE WORLD HEALTH ORGANIZATION. (Eng.) Anthony, P. P.; Ishak, K. G.; Kayak, N. C.; Poulsen, H. E.; Scheuer, P. J.; Sobin, L. H. (Middlesex Hosp. Medical Sch., London, England). *J. Clin. Pathol.* 31(5):395-414; 1978.

Guidelines on the definition, nomenclature, and classification of cirrhosis, chronic hepatitis, and hepatic fibrosis are presented, taking into account

comments and criticisms from a number of pathologists and hepatologists throughout the world. Cirrhosis is defined as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules. The complete classification of cirrhosis in an individual case would take into account its morphological features, etiology, stage of evolution, activity, and complications. Among the systems of morphological categories currently in use, the division of cirrhosis into micronodular and macronodular forms is preferred. The former division is described as a cirrhotic liver in which nearly all nodules are less than 3 mm in diameter, whereas the latter is described as having many nodules over 3 mm. When micro- and macronodules are present in approximately equal proportions, the term "mixed" may be applied. Etiological diagnosis is usually reached by a combination of epidemiological, clinical, biochemical, immunological, and histological investigations. Established etiological associations include viral hepatitis, alcoholism, metabolic disorders, biliary disease, venous outflow obstruction, toxins and therapeutic drugs, and intestinal bypass surgery; debatable factors include autoimmunity, mycotoxins, schistosomiasis, and malnutrition. Types of cirrhosis of unknown etiology include Indian childhood cirrhosis, which has a well-defined pattern, and "cryptogenic" cirrhosis, which does not. Cirrhosis is easily identified at necropsy, but may be diagnosed by surgical wedge biopsy or needle biopsy. Helpful features for the recognition of cirrhosis in a needle biopsy specimen include presence of parenchymal nodules, differences in liver cell size and appearance, fragmentation of the biopsy specimen, fibrous septa, and altered architecture and vascular relationships. Complications of cirrhosis include ischemic necrosis, siderosis, biliary obstruction, and infection. Hepatocellular carcinoma in cirrhosis is often associated with liver cell dysplasia. Chronic hepatitis is defined as liver inflammation continuing without improvement for at least 6 months, and may be classified morphologically as chronic persistent or chronic active. Hepatic fibrosis is defined as the presence of excess collagen due to new fiber formation and is classified morphologically according to its location. In addition to the causes of cirrhosis, the following causes of hepatic fibrosis are recognized: congenital, metabolic, inflammatory, parasitic, toxic, vascular, and physical.

8520 CHRONIC NORMOPROTEINEMIC HEPATOPATHY. (Ita.) Buzzelli, G.; Gallerini, L.; Guarducci, A. M.; Laffi, G.; Mazzanti, R.; Smorlesi, C.; *et al.* (Istituto di Clinica Medica, Universita, Florence, Italy). *Recent. Prog. Med. (Roma)* 63(5):497-526; 1977.

The literature on normal serum protein levels in patients with chronic hepatopathy is reviewed, and data from a series of 8/121 patients with liver cirrhosis and 13/170 with chronic active hepatitis, all of whom had normal serum protein profiles, are reported. Diagnoses were based on clinical and laboratory findings, radiology, scintigraphy, and liver biopsies. With respect to cirrhosis, a comparison of patients with normal (6.55 ± 0.15 g/dl) and abnormal (6.49 ± 0.14 g/dl) serum protein levels revealed a statistically significant difference in the BSP retention test ($p<0.01$), in serum albumin level (3.81 ± 0.13 versus 2.99 ± 0.15 g/dl, resp., $p<0.01$), and in the levels of serum IgA (233.6 ± 51.4 versus 433.5 ± 67.2 mg/dl, resp., $p<0.05$), IgG (1,357.1 ± 132.5 versus 1,840 ± 143.8 mg/dl, resp., $p<0.05$), and IgM (127.9 ± 12.1 versus 262.4 ± 45.3 mg/dl, resp., $p<0.02$). Patients with cirrhosis and normal serum protein levels had normal protein electrophoretic patterns and normal serum levels of immunoglobulins. In chronic active hepatits, significant differences were found between patients with normal and with abnormal blood protein levels with respect to the total protein level (6.78 ± 0.12 versus 7.32 ± 0.14 g/dl, resp., $p<0.01$) and the IgG level (1,367.5 ± 124.5 versus 2,334.1 ± 193.4 mg/dl, resp., $p<0.01$). The antistreptolysin levels were elevated in patients with chronic active hepatitis and abnormal serum protein level ($p<0.01$). Follow-up of six of eight patients with liver cirrhosis and normal serum protein levels showed that one had retained a normal level for 1 yr, two for 18 months, two for 2 yr, and one for 3 yr. Follow-up of 13/13 patients with chronic active hepatitis and normal serum protein levels revealed that 6 continued to have normal levels for 6 months, 4 for 1 yr, 2 for 18 months, and 1 for 2 yr.

8521 TOPOLOGICAL ANALYSIS OF THE MORPHOGENESIS OF LIVER CIRRHOSIS. (Eng.) Takahashi, T. (Tohoku Univ. Sch. Medicine, Seiryomachi, Sendai, Japan). *Virchows Arch.* [*Pathol. Anat.*] 377(3):189-201; 1978.

Topological analysis of the morphogenesis of liver cirrhosis was carried out, using the total connectivity number $p1$, in the livers of six autopsy cases of chronic liver disease. The total p_1 amounted to 6,100,000 in chronic hepatitis of the periportal type and to 6,350,000 in portal cirrhosis, but to only 100,000 in posthepatic cirrhosis of the coarse-nodular type. In view of the similarity of values for p_1 in chronic hepatitis and portal cirrhosis, the former is considered to give rise to the latter by continuous change in structure or through "piecemeal" progression of the periportal lesion. Development of posthepatitic cirrhosis from chronic hepatitis is possible only through a substantial reduction in p_1, which is brought about by parenchymal necrosis that is extensive enough to cause multiple disconnection of the network.

8522 METABOLISM OF CARBOHYDRATES, ADMINISTERED BY THE PARENTERAL ROUTE, IN PATIENTS WITH CIRRHOSIS OF THE LIVER. (Eng.) Brodanova, M.; Brodan, V.; Filip, J.; Kordac, V.; Marecek, Z.; Kuhn, E. (Faculty General Medicine, Charles Univ., Prague, Czechoslovakia). *Acta Univ. Carol.* [*Med.*] *(Praha)* 23(1/2):17-129; 1977.

A study was conducted to evaluate metabolic response to the parenteral administration of simple carbohydrates in cirrhotic patients, to determine the effect of tolbutamide and biguanide on this metabolism, and to determine if the sugars were suitable for the treatment of patients with liver cirrhosis. Groups of subjects, selected from 65 patients with morphologically confirmed liver cirrhosis, received the following quantities of sugars and/or antidiabetic agents: a 50% infusion (500 ml of 10% solution) of glucose (G) over 30 min, 24 mg fructose (F) i.v. in a 20% solution over 6 min, 50 g sucrose (S) i.v. as a 10% solution over 10 min, 50 g F or 50 g xylitol (X) as a 10% solution over 30 min, each sugar alone or after the i.v. administration of 1 g tolbutamide (T) or after the administration of 260 mg/day of butyl biguanide for 1 week. Measurements of blood sugar, lactate (LA), pyruvate (PY), immunoreactive serum insulin (IRI), fructose, uric acid, and ammonia levels were made in samples of venous blood collected during infusion and for 45 min after infusion. Similar measurements on healthy controls were available for comparison. The blood sugar level rose after G, declined slightly after S and X, and was unchanged after F. T hastened the decline in blood sugar level after G, and in the other sugars it led to a decline of blood sugar below the initial level. B slightly delayed the disappearance of blood sugar after G, and produced a permanent rise in blood sugar level after S. Serum IRI was highest after G, followed by F, X, and S. Tolbutamide potentiated the IRI response during infusion and during the subsequent 45 min. B reduced the IRI increments after all sugars except F. The LA increments were greatest after F, slightly reduced by T, and increased by B. PY changes after F were always positive, and after X, always negative. T and B gave a similar order of change. The LA:PY ratio increased after all sugars and was dependent on the amount of sugar. It was highest after F and lowest after G. T, and thus insulin, enhanced the sugar metabolism and gave ties of retarding the release of gastrin from G cells and of inhibiting the action of gastrin on parietal cells, it may be difficult to reveal a quantitative relationship between the gastric G cell and parietal cell populations.

8523 THE EFFECT OF GLUCAGON ON FREE PLASMA AMINO ACIDS IN CIRRHOTICS AND HEALTHY CONTROLS. (Eng.) Brodan, V.; Brodanova, M.; Andel, M.; Kuhn, E. (Inst. Clinical and Experimental Medicine, Budejovicka 800, Prague 4-Krc, PSC 14622, Czechoslovakia). *Acta Hepatogastroenterol. (Stuttg.)* 25(1):23-29; 1978.

Initial plasma amino acid levels and their changes after glucagon administration (1 mg by infusion) were studied in seven patients with compensated liver cirrhosis and seven healthy controls after an overnight fast at absolute bedrest. Cirrhotic patients had significantly elevated basal levels of tyrosine, citrulline, tryptophan ($p<0.01$), threonine, phenylalanine, and methionine ($p<0.05$) and significantly lower levels of serine and ornithine ($p<0.05$). Total amino acidemia was significantly higher ($p<0.01$) in the cirrhotics (3.353 ± 0.234 mM) than in controls (2.978 ± 0.167 mM). Glucagon administration caused significant decreases in the levels of ornithine, leucine, lysine, histidine, arginine, methionine ($p<0.01$), alanine, glycine, valine, proline, glutamate, and citrulline ($p<0.05$). The initial concentrations of amino acids exhibited a negative correlation with the change in their concentrations after glucagon both in the healthy controls ($r=-0.766$, $p<0.001$) and in the cirrhotics ($r=0.591$, $p<0.001$); hence, the decrease after glucagon is a function of the initial concentration. Excluding cystine-cysteine (which increased) the mean percentage decline in total amino acid level was -14.57 ± 4.84% in controls and only -9.14 ± 5.85% in cirrhotics after glucagon, the difference between the two groups being significant ($p<0.01$). The percentage decrease in essential amino acids in each group was nearly the same. Serum ammonia levels increased significantly in both groups ($p<0.05$), while the serum urea level increased significantly ($p<0.01$) only in the healthy controls.

8524 HYDROELECTROLYTIC METABOLISM IN LIVER CIRRHOSIS: PHYSIOPATHOLOGICAL AND THERAPEUTIC ASPECTS OF THE REINFUSION OF CONCENTRATED ASCITIC FLUID. (Ita.) Borghetti, A.; Fiaccadori, F.; Bruschi, G.; Coruzzi, A.; Guariglia, A.; Ghinelli, F.; *et al.* (Istituto di Semeiotica Medica, Universita degli Studi di Parma, Parma, Italy). *Minerva Med.* 69(1):53-66; 1978.

The acute effects of the reinfusion of concentrated ascitic fluid was determined in nine cirrhotic patients. During basal conditions, the plasma volume and the pools of the Na^+ and K^+ were evaluated in relation to total water volume with radioisotopic techniques. During reinfusion (10-12 hr), the elimination of Na^+, K^+, and H^+; the osmolar clearance; and the creatinine clearance were evaluated. During basal conditions, there were no significant variations in the blood concentrations of Na^+ and K^+ or in the osmolarity of the blood; the concentration of albumin was markedly low; the total body water was markedly elevated; the plasma volume and the sodium pool were augmented; the potassium pool was diminished; the ratio of plasma volume to the sodium pool was reduced; and renal function was reduced with respect to glomerular filtration. During the extraction-reinfusion treatment, there was no evidence of significant modifications of arterial pressure; the plasma volume was increased by 28%; the clearance of creatinine was significantly increased; and diuresis increased with respect to that in the pretreatment control period. After the treatment, the plasma volume remained elevated; the renal functions corresponded to those during basal conditions; and the volume of diuresis was reduced and returned (as in the period of pretreatment) to hypertonic conditions. These results should be taken into consideration when treating the ascitic syndrome.

8525 ALKALINE PHOSPHATASE ISOENZYMES IN LIVER CIRRHOSIS. (Eng.) Burlina, A.; Bugiardini, R. (Centro Ospedaliero Borgo Trento, I-37100 Verona, Italy). *Enzyme* 23(2):121-126; 1978.

The origin and diagnostic and prognostic significance of the slow fraction (ALP_4) of alkaline phosphatase found in liver cirrhosis were investigated in sera from 114 patients with liver diseases. The isoenzymatic tracings of sera from 18 of the 49 patients with cirrhosis showed the ALP_4 band. In the presence of 5×10^{-3} M L-phenylalanine, sera with the ALP_4 band showed a mean residual phosphatase activity of 63.4-70.5% of the original activity, whereas sera without this band showed a mean residual phosphatase activity ranging from 74.3 to 85%. After heating sera without the ALP_4 band at 56 C for 10 min, normophosphatemic sera showed normal residual activity; hyperphosphatemic sera were notably thermoresistant, with residual activity between 30% and 52%. The sera with the ALP_4 band were thermolabile, with a residual activity ranging between 11% and 34.5%.

8526 EFFECTS OF MEDROXYPROGESTERONE ON THE LIVER FUNCTION AND DRUG METABOLISM OF PATIENTS WITH PRIMARY BILIARY CIRRHOSIS AND CHRONIC ACTIVE HEPATITIS. (Eng.) Sotaniemi, E. A.; Hynynen, T.; Ahlqvist, J.; Ahokas, J. T.; Puoskari, U.; Pelkonen, I. (Clinical Res. Unit, Univ. Oulu, SF-90220 Oulu 22, Finland). *J. Med.* 9(2):117-128; 1978.

Medroxyprogesterone acetate, a synthetic progestin, was administered to six patients with primary biliary cirrhosis (PBC) and chronic active hepatitis (CAH) to evaluate its therapeutic effect through analysis of liver function and drug metabolism. Before this trial, all patients except one had been treated with prednisolone, azathioprine, and/or phenobarbitol and had developed resistance and/or side-effects to these drugs. During treatment with doses of 10-15 mg medroxyprogesterone for periods of 8-76 months, liver function tended to normalize. Significant reductions in the levels of total serum bilirubin (from 29.0 ± 31.0 to 14.0 ± 14.8 μml/l, $p<0.05$), serum alkaline phosphatase (from 503.7 ± 411.8 to 369.5 ± 275.2 U/l, $p<0.05$), and BSP retention (from 27.4 ± 11.2 to 12.5 ± 10.2%, $p<0.03$) were noted. The levels of serum antibodies declined and the hepatic metabolic ability, as tested by serum albumin levels and indices of drug metabolism, improved. Serum albumin level rose from 41.7 ± 2.3 to 43.3 ± 2.8 g/l, $p<0.01$, plasma antipyrine half-life was reduced from 15.1 ± 7.1 to 7.7 ± 0.7 hr, $p<0.05$, and the cytochrome P-450 content of biopsy samples rose from 6.8 ± 1.5 to 10.5 ± 5.8 nmol/g during treatment. The subjective symptoms of the diseases decreased. The authors believe that the improved effect on the clinical condition of the patients may be due to the immunosuppressive effect of medroxyprogesterone compared with barbiturate treatment. The study shows that medroxyprogesterone treatment of patients suffering from PBC or CAH leads to the suppression of the immunological manifestations of the disease and may be a valuable alternative for patients who have developed resistance or undesirable reactions to previous therapy.

8527 SOMATOMEDIN IN CHRONIC LIVER DISEASE. (Eng.) Marek, J.; Schullerova, M.; Schreiberova, O. (Third Medical Clinic, Charles Univ., Prague, Czechoslovakia). *Rev. Czech. Med.* 22(4):194-201; 1976.

Since recent studies have suggested that the liver is an important site of somatomedin formation, serum somatomedin levels were determined in 6 patients with cirrhosis, 3 patients with chronic hepatitis, and 13 healthy controls. The somatomedin level in each patient was markedly lower than the mean level in the healthy subjects. There was a significant correlation between the serum albumin levels and the somatomedin level in the patients with liver disease ($r=0.8653$, $p<0.01$). These results are consistent with the results of previous studies indicating that the liver is an important site of somatomedin synthesis.

8528 FORMATION OF VITAMIN D METABOLITES FROM ^{3}H- AND ^{14}C-RADIOLABELLED VITAMIN D-3 IN CHRONIC LIVER DISEASES. (Eng.) Long, R. G.; Skinner, R. K.; Wills, M. R.; Sherlock, S. (Royal Free Hosp., Pond St., London NW3 2QG, England). *Clin. Chim. Acta* 85(3):311-317; 1978.

The formation of 25-hydroxy vitamin D (25-OHD) and dihydroxyvitamin D metabolites was studied using a double-isotope method. To evaluate the hypothesis that there is a failure to form dihydroxyvitamin D metabolites in liver disease. Eight patients were studied; seven had primary biliary cirrhosis, and the eighth had cirrhosis due to chronic active hepatitis. Four were vitamin D replete, and four were vitamin D depleted, as judged by the serum 25-OHD concentrations (<4.15% of 25-OHD dose/l serum). Three of the four vitamin D-depleted patients (including 2 with histological osteomalacia) formed radioactive 1,25-dihydroxycholecalciferol; one of the patients with a normal serum 25-OHD concentration also synthesized 1,25-dihydroxycholecalciferol, the mean maximum serum level being 4.03% of the dose/l serum in the four patients. 24,25-Dihydroxycholecalciferol was formed by all vitamin D-replete patients but not by any of the patients who were vitamin D depleted. 25,26-Dihydroxycholecalciferol was formed by all patients, except one who also formed no 1,25-dihydroxycholecalciferol. This study suggests that patients with liver disease can form dihydroxy vitamin D metabolites.

8529 THE CIRRHOTIC PATIENT AND THE PORTACAVAL SHUNT IN 1977. (Fre.) Huet, P. M.; Marleau, D.; Lavoie, P.; Tanguay, S.; Viallet, A. (Hopital Saint-Luc, 1058, rue St. Denis, Montreal, Quebec H2X 3J4, Canada). *Rev. Fr. Gastroenterol.* (131):29-36; 1977.

The results of portacaval shunts were evaluated in 30 patients in whom the procedure was performed because of liver cirrhosis and hemorrhage from esopha-

geal varices. Ten patients also underwent arterialization of the portal vein. Prior to surgery, the functional portal blood supply to the liver was evaluated by the uptake of indocyanine green (ICG) by the hepatocytes or of ^{125}I-albumin by the Kupffer cells. Of the 20 patients who had only the portacaval shunt, 19 survived 1-50 months (average, 18 months). Hepatic encephalopathy developed in 7/19. All of the patients who developed encephalopathy had a minimal decrease in portal blood supply, with ICG uptake values above 20% (average, 33.7%). The 12 patients who did not develop encephalopathy had ICG uptake values below 20% (average, 11%). Of the 10 patients who had arterialization in addition to the shunt, 7 survived 12-50 months (average, 24.3 months), and none developed encephalopathy. The values for ICG uptake were low in three of seven patients and over 20% (average, 31.9%) in four of seven patients. This study suggests several conclusions: (1) all patients who undergo portacaval shunts should have prior studies of liver hemodynamics, (2) patients with depressed functional portal blood supply are less likely to develop hepatic encephalopathy than are those with normal supply, and (3) patients with a minimal decrease in portal blood supply appear to benefit from arterialization in addition to the shunt, although further evidence for this conclusion is needed.

8530 COINCIDENCE POSITRON IMAGING OF THE LIVER AND HEART FOLLOWING RECTAL ADMINISTRATION OF ^{13}N-AMMONIA IN LIVER CIRRHOSIS. (Eng.) Fukuda, N.; Tateno, Y.; Rikitake, T.; Matsumoto, T.; Tomitani, T.; Suzuki, K.; *et al.* (Natl. Inst. Radiological Sciences, Anagawa, Chiba-shi 280, Japan). *Radioisotopes* 26(12):872-875; 1977.

8531 HEPATIC CIRRHOSIS IN VARANASI (A HAEMOSTATIC APPRAISAL). (Eng.) Singh, D. S.; Dube, B.; Gupta, J. P. (JIPMER, Pondicherry, India). *J. Indian Med. Assoc.* 69(2):26-30; 1977.

See also, 8144, 8152, 8179, 8297, 8311, 8439, 8441, 8442, 8443, 8444, 8450, 8455, 8459, 8460, 8461, 8465, 8466, 8467, 8481, 8487, 8508, 8513, 8515, 8518.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

8532 SURGICAL PROBLEMS OF PRIMARY SCLEROSING CHOLANGITIS. (Fre.) Salembier, Y.; Dupont, A.; Bleuez, M.; Paris, J. (Cite Hospitaliere, 69, rue de la Louviere, 59000 Lille, France). *J. Chir. (Paris)* 114(6):451-460; 1977.

The clinical symptoms, pathology, differential diagnosis, and treatment of primary sclerosing cholangitis are discussed and five cases are reported. The cases included three women (ages 22, 21, and 43 yr, resp.) and two men (ages 53 and 43 yr, resp.). Two of the patients had typical symptoms such as icterus, pruritus, bile retention with enlarged interlobular hepatic ducts, and hepatomegaly. The condition, described for the first time in 1924, is characterized by a diffuse inflammation with fibrous thickening and stenosing of the biliary ducts in the absence of lithiasis. Primary sclerosing cholangitis affects men primarily (62.5% of cases), whereas primary biliary cirrhosis affects women primarily (75% of cases). Additionally, in the former entity, there are no antimitochondrial antibodies or xanthoma present, and pruritus is not a constant symptom (present in only 1/4 of the cases). Microscopic examination of the liver shows the biliary canals surrounded by a circular fibrosis. Submucosal and subserosal fibrosis with inflammatory infiltration is always observed in the extrahepatic ducts in primary sclerosing cholangitis and may only be observed there in some cases of primary biliary cirrhosis. Ulcerative colitis is associated with primary sclerosing cholangitis in about one of three patients. Treatment of choice is corticoids, specifically prednisolone, 10-25 mg/day, alone or with biliary drainage to relieve cholestasis. A biliary-gastrointestinal bypass may be performed at the convergence of the biliary ducts, but the surgery would deprive the patient of local treatment such as injection of cortisone into the biliary drain. Prognosis remains poor with average survival 4 yr from time of definitive diagnosis.

8533 INVOLVEMENT OF THE PANCREAS IN ACUTE CHOLECYSTITIS. (Ger.) Brunner, H.; Neher, M.; Schmidt, H. D. (Chirurgische Universitatsklinik, Langenbeckstrasse 1, 6500 Mainz, W. Germany). *Therapiewoche* 28(8):1419, 1422; 1978.

Involvement of the pancreas was found in 21 of 72 patients with acute cholecystitis. The pancreatic involvement was identified as an enzymatic shift without pathological substrate in the pancreas in

nine cases, as accompanying pancreatitis in eight, as edematous pancreatitis in one, and as hemorrhagic necrotizing pancreatitis in three. In another series of 97 patients with pancreatitis (33 cases of edematous and 64 cases of hemorrhagic necrotizing pancreatitis), acute cholecystitis was found in 4 cases (1 in edematous and 3 in hemorrhagic necrotizing pancreatitis). Even though pancreatic involvement is rather frequent in choledocholithiasis, the pancreatitis in such cases is usually of canalicular genesis.

8534 THE PREREQUISITES FOR LOCAL LYSOLECITHIN FORMATION IN THE HUMAN GALLBLADDER. II. STUDIES ON THE POSITIONAL SPECIFICITY OF THE PHOSPHOLIPASE A ACTIVITY. (Eng.) Tagesson, C.; Norby, S.; Sjodahl, R. (Dept. Medical Microbiology, Univ. Linkoping, S-581 85 Linkoping, Sweden). *Scand. J. Gastroenterol.* 13(4):417-422; 1978.

The positional specificity of phospholipase A in human gallbladder epithelium was studied using biosynthetically radiolabeled diacylphosphoglycerides as substrates. When homogenized epithelial cells were incubated with ^{14}C-palmitate-labeled *Escherichia coli*, ^{14}C-phosphatidylethanolamine (^{14}C-PE) was degraded. During this degradation, ^{14}C-lyso-PE and ^{14}C-fatty acids were formed. When the homogenized cells were incubated with ^{14}C-oleate-labeled bacteria, ^{14}C-PE was degraded, but no ^{14}C-lyso-PE was formed. Instead, the formation of ^{14}C-fatty acids was increased. The pH optimum of ^{14}C-fatty acid synthesis with ^{14}C-oleate- or ^{14}C-palmitate-labeled bacteria was 7.0. Since ^{14}C-palmitate occupies predominantly the 1-acyl position and ^{14}C-oleate the 2-acyl position of the major *E. coli* diacylphosphoglycerides, these findings suggest that (a) the phospholipase attacks the 2-position of diacylphosphoglycerides and (b) a complete deacylation of diacylphosphoglycerides in the gallbladder wall is brought about by the combined action of phospholipase A_2 and a lysophospholipase, the latter having the ability to hydrolyze the 1-acyl-lysophosphoglyceride. It appears that the biochemical prerequisites for a local formation and degradation of lysolecithin in the gallbladder are met by the positional specificity of the enzymes present. This finding substantiates the hypothesis that lysolecithin is an adjustable mediator of aseptic cholecystitis.

8535 ENDOSCOPIC TRANSPAPILLARY SPLITTING OF A CHOLEDOCHOCELE. (Ger.) Miederer, S. E.; Lindstaedt, H.; Siedek, M.; Franken, Th. (Medizinische Poliklinik, Universitat Bonn, Wilhelmstr. 35, 5300 Bonn, W. Germany). *Dtsch. Med. Wochenschr.* 103(5):216-219; 1978.

A choledochocele with bile outflow obstruction from the biliary and pancreatic ducts was diagnosed by endoscopic retrograde cholangiopancreaticography in a 67-yr-old man who presented with radiating upper abdominal pain and a constantly elevated gamma-glutamyltransferase level 16 yr after cholecystectomy for cholecystolithiasis. After distention of the choledochocele with contrast medium, a papillotome was introduced through the papilla, and the choledochocele was split open. The patient required neither anesthesia nor an abdominal operation. There were no complications, and the patient experienced immediate complete cessation of the pains. The bile flow from both systems became normal.

8536 GRAY-SCALE ULTRASOUND B-SCANNING IN CASE OF CHOLELITHIASIS, ESPECIALLY WITH NEGATIVE CHOLECYSTORADIOGRAM. COMPARISON WITH SURGICAL AND RADIOGRAPHIC FINDINGS. (Ger.) Koppers, B. (Institut fur Rontgendiagnostik, Technische Universitat Munchen, Westendstrasse 85, D-8000 Munich 2, W. Germany). *Med. Klin.* 73(8):279-283; 1978.

The results of gray-scale ultrasound B-scanning were compared with those of surgery and cholecystoradiography in 37 patients. The gallbladder was visualized by ultrasonography in 36/37 patients. Surgery or radiography revealed no stones in 16 cases; the ultrasonic scan was correctly negative in 14/16 cases and falsely positive in 2/16. Surgery or radiography revealed gallstones in 20 cases; the sonography was correctly positive in 16/20 cases and falsely negative in 4/20. The correlations between ultrasound and the other techniques were significant ($p<0.001$). The findings indicate the great diagnostic value of sonography, especially in the case of neagtive radiography.

8537 ENDOSCOPIC PAPILLOTOMY AND REMOVAL OF GALLSTONES. (Eng.) Seifert, E. (Staedt Krankenhaus Kemperhof, 1st Med. Klin., Koblenzer Str. 115, 5400 Koblenz, W. Germany). *Am. J. Gastroenterol.* 69(2):154-159; 1978.

The results, complications, and risks of endoscopic papillotomy (EPT) in the treatment of choledocholithiasis or papillary stenosis in a total of 995 patients in nine centers in Germany are reported. The procedure was successful in 880 of the 955 cases. Choledocholithiasis was the most frequent indication for EPT, being present in 743 patients. Seventy-seven percent of these 743 patients were cholecystectomized. Papillary stenosis was the indication for EPT in 14.7% of the cases and tumor in 1.3%. Choledocholithiasis was successfully treated by EPT (either by spontaneous delivery or by extraction) in 85.9% of cases. Complications developed in 7.3% of the 880 cases. These included perforation (13 cases), hemorrhage (16), cholangitis (13), pancreatitis (15), and stone impaction following the procedure (6). In the remaining case, the Dormia basket became impacted into the common bile duct and had to be removed surgically. Most complications occurred within 24 hr of the procedure, only 12% occurring on the 2nd day. Sixty-five percent of the complications were treated conservatively, 5% endoscopically, and 30% surgically. Most hemorrhages were stopped conservatively, and 9 of 13 perforations could be treated in this manner. Cholangitis was reduced by the introduction of an indwelling tube in the common duct immediately after EPT. All but one case of

pancreatitis was treated conservatively. The mortality rate was 1.7%, and this was reduced to 1.25% when patients who died of cardiac failure were excluded. The remaining causes of death included hemorrhage (1 case), septicemia (3), liver abscesses (1), pancreatitis (3), peritonitis (1), and cholangitis (1). It is concluded that EPT is useful in the treatment of high-risk but operable patients with choledocholithiasis or papillary stenosis.

8538 A FOLLOW-UP STUDY OF LIVER FUNCTION TESTS AFTER SURGICAL TREATMENT FOR GALLSTONE DISEASES. (Eng.) Harada, T.; Sugaya, H.; Maehara, M.; Kimura, K.; Hisauchi, T.; Tajima, Y.; *et al.* (Dokkyo Univ. Sch. Medicine, Mibu, Tochigi, 321-02 Japan). *Gastroenterol. Jpn.* 12(6):446-454; 1977.

Alterations in liver function as measured by SGOT, SGPT, serum alkaline phosphatase, and serum bilirubin levels were studied in 204 patients during the 1st month and in 113 patients up to 1 yr after surgery for gallstone disease. Postoperative prognosis and subjective symptoms were collected by a questionnaire mailed to 201 patients 10 months to 5 yr after surgery. At 1 month of follow-up, 204 patients were separated into five groups: A, 104 patients, liver function normal before and after surgery; B, 47 patients, liver function improved after surgery; C, 14 patients, liver function transiently aggravated after surgery but later improved; D, 14 patients, liver function abnormal before and after surgery; and E, 25 patients, liver function obviously aggravated postoperatively. At 1 yr of follow-up, 113 patients were distributed in these five groups as follows: 69, 29, 20, 3, and 12 in A, B, C, D, and E, resp. In the patients studied from 1 month to 1 yr after surgery, the incidence of liver dysfunction (patients classified into groups D and E) was higher in cases of choledocholithiasis (28.6%) than in cases of cholecystolithiasis (12.0%); it was also higher in cases operated by choledochotomy (17.6%) and with T-tube drainage (21.5%) than in cases operated by cholecystectomy (8.8%), and in cases requiring blood transfusion (22.3%) than in cases without transfusion (12.0%). The effects of blood transfusion and the presence of pathologic conditions requiring transfusion contributed to the high incidence of abnormal liver function in the 18 cases with blood transfusion. The presence of acute biliary infection requiring use of antibiotics also produced a higher incidence of liver dysfunction. The average operation time for patients in group A was 119.4 ± 43.1 min; this time tended to be longer in the other groups (184.1 ± 91.5 min in B and 163.9 ± 45.1 min in E). Thus, the location of gallstones, the surgical technique used, the operation time, and the need for blood transfusion and for antibiotics were found to influence the liver function parameters. The postoperative survey revealed a favorable prognosis in 73.2%, relatively favorable in 17.1%, slightly poor in 8.0%, and poor in 1.2% of the 164 patients replying to the questionnaire. Subjective symptoms reported diarrhea (25.9%), back pain (25.9%), and abdominal pain (22.1%).

8539 DIET AND CHOLESTEROL GALLSTONES: A MULTICENTER STUDY. (Eng.) Sarles, H.; Gerolami, A.; Cros, R. C. (Unite de Recherches de Pathologie Digestive U-31 Inserm, 46, chemin de la Gaye, F-13009 Marseille, France). *Digestion* 17(2): 121-127; 1978.

The role of diet in the development of cholesterol gallstones was studied by comparing changes in mortality due to gallstones with changes in diet during recent years in the southeast of France, and by comparing diet with the prevalence of gallstones in seven countries. Statistics in France obtained between 1939 and 1971 show that total calorie, lipid, and animal protein intake increased markedly from 1943 to 1967. The onset of an increase in mortality due to gallstone disease began 6 to 7 yr after the onset of the dietary increases. A comparison of the incidence of gallstones found at autopsy in France, India, Japan, Portugal, South Africa, Sweden, and Uganda with the types of diets in these countries revealed that low-calorie, low-protein, low-lipid, high-vegetable content diets are associated with low incidences of gallstones. A positive correlation between calorie intake and gallstone incidence was found for calorie intakes lower than 3,000 kcal/day, whereas no such association was found for higher calorie intakes because incidence of gallstones then varies greatly. The results suggest that a hypocaloric diet associated with a low intake of fat and protein is protective against cholelithiasis.

8540 DIET AND CHOLESTEROL GALLSTONES: A FURTHER STUDY. (Eng.) Sarles, H.; Gerolami, A.; Bord, A. (Unite de Recherches de Pathologie Digestive U 31 Inserm, 46, chemin de la Gaye, F-13009 Marseille, France). *Digestion* 17(2):128-134; 1978.

The diets of two groups of women with cholesterol gallstones were compared with those of two groups of women without gallstones, to determine whether dietary factors influence the development of cholelithiasis. The total caloric intake, caloric intake excluding alcohol, carbohydrate intake, animal protein intake, vegetable protein intake, total protein intake, and fat intake in 202 women without gallstones were similar to those in 11 women with cholesterol gallstones. A similar study in 50 patients and 50 controls also showed no significant differences in the dietary intake in the two groups. The results of the present studies do not support those of studies following World War II, in which increased caloric intake after the war was followed by an increased incidence of cholelithiasis. It is proposed that undernutrition, as seen during the war, may reduce the chance of cholelithiasis in women.

8541 IN-VITRO STUDIES OF GALLSTONE DISSOLUTION: THE EFFECT OF ADDED HEPARIN IN BILE SALT SOLUTIONS. (Eng.) Furnival, C. M.; Burnett, W.; Green, M. K.; Selvage, N. C.; Cavaye, G.; Hardie, I. R. (Clinical Sciences Building, Royal Brisbane

Hosp., Brisbane, Queensland 4020, Australia). *Aust. N.Z. J. Surg.* 47(6):828-831; 1977.

The dissolving effect of four bile salt solutions upon gallstones was tested *in vitro* using syphonage to continuously fill and empty test chambers for a 10-day period. Solutions of sodium cholate (SC), sodium taurocholate (ST), sodium deoxycholate (SD), and sodium chenodeoxycholate (SCD), all 200 mM, were tested, and physiological saline (S, 0.9%), heparinized saline (HS, 100,000 IU/l), and lecithin (L, 100 mM) were used as controls; 226 stones from 38 patients were tested. No stones exposed to S, HS, or L lost weight. All stones exposed to bile salts lost weight (26% in SC, 26% in ST, 52% in SD, and 41% in SCD); the weight loss in SD was significantly greater than that in SC or ST ($p<0.01$). A combined solution of SC and ST produced a greater weight loss (38%) than did the components alone, but still less than did SD. The weight loss in SCD was significantly greater than that in ST or SC ($p<0.001$). Addition of H to solutions of SC or SD enhanced the weight loss of stones to 43% ($p<0.01$) or 77% ($p<0.05$), resp.; this enhancing effect persisted ($p<0.01$) when weight-matched stones from each of 10 patients were tested in the two solutions: in 9/10 cases, the H-enhancing effect on weight loss occurred. The addition of L to SC, SD, or SCD solutions produced a significant increase in weight loss only in the case of SC (36%, $p<0.05$), with slight increases in the other tests. Fragmentation occurred in 34 stones, with the greatest incidence in SD + H solution. The results suggest that the optimal solution for clinical use is a combination of SC and H because the possible toxic side-effects of SD + H preclude its use.

8542 CLINICAL STUDIES ON DISSOLUTION OF GALLSTONES USING URSODEOXYCHOLIC ACID. (Eng.) Okumura, M.; Tanikawa, K.; Chuman, Y.; Koji, T.; Nakawa, S.; Nakamura, Y.; *et al.* (Fukuoka Univ. Sch. Medicine, 34 Nanakuma, Nishi-ku, Fukuoka, 814 Japan). *Gastroenterol. Jpn.* 12(6):469-475; 1977.

The clinical usefulness of ursodeoxycholic acid (UDCA) in the dissolution of gallstones was investigated in 74 patients with gallstones in functioning gallbladders who were treated for 6 months or more (mean duration of treatment, 8.5 months). All patients received 450 mg of UDCA daily. During the study, other choleretics, anticholesterol drugs, and thyroid hormones were not used. The size and number of gallstones were recorded by cholecystography, the criterion for drug efficacy being a reduction in stone size of at least 20% in diameter compared with diameter before treatment. The gallstones completely disappeared in 16 patients and partially dissolved in 16 others (effective rate, 43.2%). The gallstones remained unchanged in 35 patients and required surgery in 7. In 26 of 35 cases with gallstone symptoms, a marked reduction in the frequency and severity of symptoms was noted. UDCA effectiveness for total dissolution was significantly higher with smaller stones (33.3%, 18.2%, and 5.9% for stones <5, 6-10, and >11 mm, resp.) Large numbers of small-sized stones were most easily reduced, the effective rate for complete or partial dissolution being 21.4% for small (<2 mm), 24.0% for moderate (3-9 mm), and 38.1% for large (>10 mm) numbers of gallstones. The rate of complete disappearance in 24 patients with floating gallstones was 33.3% and 19.1% in 42 without floating gallstones. The effective rate for complete or partial dissolution was 27.3% among 22 patients with some calcification of gallstones and 52.5% among 46 patients without calcification. The disappearance rates in this study clearly exceed the rate of spontaneous disappearance of gallstones (about 7.8%). Administration of either chenodeoxycholic acid (CDCA) or UDCA produces a marked increase in excretion of UDCA in bile, diminished cholesterol saturation of bile, and ultimately, the dissolution of cholesterol gallstones. While CDCA was harmful to liver function and increased hemolytic activity due to formation of increased amounts of lithocholic acid, UDCA produced no significant changes in blood or liver function tests. UDCA appears to be as effective as CDCA in the dissolution of gallstones with few side-effects (soft stools in 4 patients and constipation in 1).

8543 MODIFICATIONS INDUCED BY URSODEOXYCHOLIC ACID (UDCA) ON THE LIPID COMPOSITION OF BILE AND PLASMA IN PATIENTS WITH CHOLESTEROL GALLSTONES. (Ita.) Salvioli, G.; Salati, R.; Lugli, R.; Fratalocchi, A. (Istituto di Clinica Medica Generale dell'Universita di Modena, Modena, Italy). *Gazz. Med. Ital.* 136(10):517-530; 1977.

The effect of ursodeoxycholic acid (UDCA) on lipid and cholesterol levels in bile and plasma and on gallstone dissolution was investigated in 18 patients with cholesterol gallstones (16 patients with gallbladder stones and 2 with common duct stones). The daily doses of UDCA used were 500 mg/g (8 patients), 600 mg/g (1), 700 mg/g (1), 800 mg/g (5), or 1,000 mg/g (3); the range was 6.6-15.6 mg/kg. The lithogenic index (LI) was determined before and after 1-3 months of treatment. The LI decreased from an average value of 2.02 before treatment to an average value of 0.99 after treatment ($p<0.001$), whereas the percentage of biliary UDCA plus chenodeoxycholic (CDCA) increased from 46.4% to 72%. There was an important difference between the patients treated with 500 mg/g UDCA and those treated with greater doses: the former group had 38.94 ± 9.76 and 31.18 ± 7.95% UDCA and CDCA, resp., whereas the latter had 47.98 ± 10.9 and 26.56 ± 6.05% UDCA and CDCA, resp., after treatment. The lithocholic acid level showed no significant alterations after treatment. Radiological examination carried out every 4 months showed gallstone disappearance in two cases and reduction in five after 8-12 months of treatment. The liver function tests were unchanged during the treatment period. Bile triglyceride levels decreased significantly ($p<0.01$) due to a reduction in very low density lipoproteins, whereas there were small increases in the phospholipid levels and in high density lipoproteins. Cholesterol levels decreased in both bile and plasma. A remarkable effect on the subjective symptoms

was noted in all patients. No side-effects were recorded in any patient.

8544 THE EFFECT OF ORAL CHENODEOXYCHOLIC ACID ON CHOLESTEROL SOLUBILITY IN HEPATIC BILE. (Eng.) Matsushiro, T.; Kobayashi, N.; Suzuki, N.; Yamauchi, H.; Sato, T. (Tohoku Univ. Sch. Medicine, Sendai 980, Japan). *Tohoku J. Exp. Med.* 124(2):187-196; 1978.

To determine the optimum dose of chenodeoxycholic acid (CDCA) in cholesterol dissolution, 14 patients who underwent cholecystectomy with T-tube drainage for cholesterol gallstone disease received 250, 750, or 1,500 mg CDCA daily for 7 days, following clamping of the T-tube. Seven patients received the 250-mg dose, while the other seven received the 750-mg dose for 7 days; the dose was increased to 1,500 mg in the latter patients, and this dose was given for a further 7 days. Hepatic bile samples were taken daily from the day before treatment through the last day of treatment, by releasing the T-tube clamp. There were no significant changes in the bile cholesterol, phospholipid, or total bile acid content after treatment in any of the three treatment groups, nor in a control group of five patients. Cholesterol solubility was calculated as (B+P)/C, where B is total bile acids, P is the phospholipid level, and C is the cholesterol level in hepatic bile. Before treatment, solubility ranged between 5.3 and 6.0 in the treatment group, and the value in controls was 6.1. In the group treated with 250 mg of CDCA, the mean value was 11.0 ± 4.0 on the 2nd day, 10.4 ± 2.8 on the 4th day, and 9.6 ± 1.3 on the 6th day ($p<0.05$ in all cases). A significant increase ($p<0.05$) in cholesterol solubility was also found in patients treated with 750 mg on the fourth day, but no increases occurred in patients given 1,500 mg or in controls. When the doses were calculated in terms of body weight, increases in solubility were slight in the three patients receiving 4.5 mg CDCA/kg or less in the 250-mg/day group, the highest increase being only 70%. In the other four cases in this group, who were receiving 4.6 mg/kg CDCA or more, the increase was over 100%. Among the patients in the 750 mg/day group, three patients receiving 15.0 mg/kg or less showed a 60% increase or lower. Those receiving over 15.0 mg/kg did not show as dramatic a response as did those in the 250 mg/day group receiving 4.6 mg/kg CDCA or more. It is concluded that the optimum dose of CDCA for increasing biliary cholesterol dissolution is between 5 and 12 mg/kg. The only side-effect of CDCA was transient SGOT and SGPT elevation in a patient receiving 750 mg daily.

8545 NONLITHIASIC CHOLECYSTOPATHY OR CHOLECYSTOSIS: CLINICAL AND STATISTICAL DATA FROM 1,027 CHOLECYSTOGRAPHIES. (Spa.) Javier Adell Alvarez, F.; Pujol Soler, R.; Mozo Pous, F.; Salord Osnes, J. (Institucion Clinica "Tres Torres," Barcelona, Spain). *Rev. Esp. Enferm. Apar. Dig.* 51(4): 381-390; 1977.

8546 PRIMARY SCLEROSING CHOLANGITIS: REPORT OF A CASE. (Fre.) Spay, G.; Donne, R.; Toure, P.; Vu, Q. D. (58 rue Lt-Colonel-Prevost, 69006 Lyon, France). *Lyon Chir.* 73(5):380-384; 1977

8547 EARLY SURGERY IN ACUTE CHOLECYSTITIS. (Rus.) Shmauss, A. K.; Vekner, V. (Friedrichshain Hosp., Berlin, E. Germany). *Khirurgiia (Mosk.)* (1):87-88; 1978.

8548 VOLEMIC CHANGES IN ACUTE CHOLECYSTITIS. (Rus.) Garibdzhanian, A. L. (Faculty Dept. Surgery, I. M. Sechenov First Moscow Medical Inst., Moscow, USSR). *Sov. Med.* (9):54-57; 1977.

8549 VASCULAR AND TISSUE CHANGES OF THE GALLBLADDER IN ACUTE CHOLECYSTITIS. (Rus.) Mukhamedov, S.; Kakharov, A. (Abuali ibn-Sino Tadzhik State Medical Inst., USSR). *Dokl. Akad. Nau Tadzh. SSR* 20(7):63-67; 1977.

8550 CHANGES IN THE STRUCTURE OF THE LIVER IN CALCULOUS CHOLECYSTITIS. (Rus.) Deenichin, P.; Protokhristov, H. (Dept. Hosp. Surgery, Faculty Medicine, Plovdiv, Bulgaria). *Khirurgiia (Mosk.)* (7):35-37; 1977.

8551 GALLSTONE ILEUS. (Spa.) Barreiro Alvarez F.; Lorenzo Garcia, M. L.; Mateo Martinez, A.; Mata Juberias, A. (Ciudad Sanitaria de la S. S. "La Paz," La Paz, Argentina). *Rev. Esp. Enferm. Apar. Dig.* 51(6):667-678; 1977.

8552 GALLSTONE ILEUS. (Ger.) Helwing, E.; Jung, D. (Medizinische Hochschule, Pasteurallee, 3000 Hannover 51, W. Germany). *Dtsch. Med. Wochenschr.* 102(51):1887-1888; 1977.

8553 DISSOLUTION OF BILIARY CALCULI BY MEANS OF CHENODEOXYCHOLIC ACID. INITIAL OBSERVATIONS. (Cze.) Hrabak, P.; Doskova, M.; Skorepa, J. Souchova, E. (IV. Interni Klinika Fakultni Nemocnice I. s poliklinikou, KUNZ SKNV, Prague, Czechoslovakia). *Cas. Lek. Cesk.* 116(43):1337-1342; 1977.

8554 THERAPEUTIC TRIAL OF DRUGS OTHER THAN CHENODEOXYCHOLIC ACID IN CHOLELITHIASIS [Abstract]. (Fre.) Capron, J. P. (Centre Hospitalier Universitaire, Amiens, France). *Gastroenterol. Clin. Biol.* 1(11):962-963; 1977.

8555 THE SHORT CHOLEDOCHUS SYNDROME: CASE REPORT AND RETROGRADE ENDOSCOPIC VISUALIZATION OF THE BILIARY SYSTEM. (Eng.) Lehmann, H.; Popken, H.; Schlaak, M. (Zentrum Konservative Medizin I der Universitat Kiel, Schittenhelmstr. 12, 2300 Kiel, W. Germany). *Acta Hepatogastroenterol. (Stuttg.)* 25(2):158-161; 1978.

LIVER AND BILIARY TRACT

8556 REVIEW OF 555 CHOLECYSTECTOMIES WITHOUT DRAINAGE. (Eng.) Carpenter, W. S.; Kambouris, A. A.; Allaben, R. D. (Mount Carmel Mercy Hosp., Detroit, MI). *Am. Surg.* 44(4):200-205; 1978.

8557 CHOLECYSTECTOMY: TO DRAIN OR NOT TO DRAIN. A RANDOMIZED PROSPECTIVE STUDY OF 200 PATIENTS. (Eng.) Maull, K. I.; Daugherty, M. E.; Shearer, G. R.; Sachatello, C. R.; Ernst, C. B.; Meeker, W. R.; Griffen, W. O., Jr. (Medical Coll. Virginia, Box 893, Richmond, VA 23298). *J. Surg. Res.* 24(4):259-263; 1978.

8558 THE PHYSICAL CHEMISTRY OF CHOLESTEROL SOLUBILITY IN BILE: RELATIONSHIP TO GALLSTONE FORMATION AND DISSOLUTION IN MAN. (Eng.) Carey, M. C.; Small, D. M. (Peter Bent Brigham Hosp., Boston, MA 02115). *J. Clin. Invest.* 61(4):998-1026; 1978.

8559 PERCUTANEOUS COMMON DUCT STONE REMOVAL. (Eng.) Carter, B. L.; Millan, V. G.; Sarno, R. C. (Tufts New England Medical Center, Boston, MA). *Abdom. Surg.* 20(3):60-65; 1978.

8560 CONTROLLED TRIAL OF CHENODEOXYCHOLIC THERAPY FOR RADIOLUCENT GALLSTONES: A MULTICENTER STUDY. (Eng.) Gerolami, A.; Sarles, H.; Brette, R.; Paraf, A.; Rautureau, J.; Debray, C.; *et al.* (Unite de Recherches de Pathologie Digestive, INSERM U-31, 46, chemin de la Gaye, F-13009 Marseille, France). *Digestion* 16(4):299-307; 1977.

8561 EXPERIENCE WITH PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY USING THE JAPANESE NEEDLE. (Eng.) Ariyama, J.; Shirakabe, H.; Ohashi, K.; Roberts, G. M. (Dept. Gastroenterology, Juntendo Univ., 3-1-3 Hongo, Bunkyo-ku, 113 Tokyo, Japan). *Gastrointest. Radiol.* 2(4):359-365; 1978.

8562 TYPE OF GALL STONES IN NORTH-WEST INDIA. (Eng.) Mooppan, M. M. U.; Jain, S. K.; Gupta, N. M.; Subrahmanyam, D.; Talwar, B. L. (Postgraduate Inst. Medical Education and Res., Chandigarh, India). *Bull. Postgrad. Inst. Med. Ed. Res. Chandigarh* 11(3):138-140; 1977.

See also, 8075, 8143, 8144, 8146, 8151, 8152, 8154, 8155, 8158, 8159, 8167, 8174, 8176, 8177, 8300, 8367, 8402, 8439, 8440, 8442, 8443, 8462, 8466, 8467, 8469, 8492, 8596, 8600.

GENERAL

8563 CARCINOEMBRYONIC ANTIGEN (CEA) TEST AND OTHER IMMUNOLOGICAL INVESTIGATIONS IN THE FOLLOW-UP OF ADENOCARCINOMA OF THE GASTROINTESTINAL TRACT. (Ger.) Hasler, E.; Spengler, A.; Berchtold, R.; Brunner, K. W.; de Weck, A. (Onkologische Abteilung, Inselspital, CH-3000 Bern, Switzerland). *Schweiz. Med. Wochenschr.* 107(47):1683-1691; 1977.

The carcinoembryonic antigen (CEA) levels were measured in 40 patients with gastric adenocarcinoma and in 85 patients with colorectal adenocarcinoma before and after surgery. The CEA level was above normal (>2.5 ng/ml) in 24/40 patients with gastric adenocarcinoma before surgery; the other 16 patients had normal CEA levels (false-negative results). Direct correlation was found between the tumor size, distant metastases, prognosis, and operability on one hand and the CEA level on the other. Radical surgery was possible in 9/16 patients with preoperatively normal CEA levels, and in 3/24 patients with elevated CEA level. Among the patients with gastric adenocarcinoma who underwent radical surgery, the incidence of recurrences has been 2/9 in the case of initially normal CEA level, versus 2/3 in the case of initially elevated CEA level. The preoperative CEA level was elevated in 58/85 patients with colorectal adenocarcinoma and normal (false-negative) in the others. Radical surgery was possible in 25/27 patients with initially normal CEA level and in 30/58 patients with elevated CEA level. The preoperatively elevated CEA level dropped to normal or decreased after surgery, depending on how radical the surgery was, while the CEA level showed further increase in the inoperable patients. The increase in the CEA level during the follow-up period was the earliest sign of distant metastases in most cases. The CEA test is a valuable tool in the follow-up of patients with gastrointestinal adenocarcinoma.

8564 NECROTISING ENTERO-COLITIS FOLLOWING UMBILICAL VEIN CATHETERISATION. (Eng.) Shah, K. J.; Corkery, J. J. (Children's Hosp.,

Ladywood Middleway, Birmingham B16 8ET, England). *Clin. Radiol.* 29(3):295-301; 1978.

Fifteen cases of neonatal necrotizing enterocolitis (NEC) following umbilical vein (UV) catheterization are reported. Ten patients had exchange blood transfusions and the others had UV infusions of bicarbonate, dextrose, or fructose for respiratory difficulties, convulsions, or hypoglycemia. Symptoms occurred from 12-72 hr after transfusion or infusion and included abdominal distention (15 patients), abdominal tenderness (9), bile-stained vomiting (9), constipation (8), blood per rectum (4), abdominal mass (2), and diarrhea (1). Twelve patients required surgery. The mortality rate was 4/15 patients. The initial diagnosis of NEC was usually made by plain radiography. Radiological features included bowel obstruction (8), peritonitis (5), ascites (4), pneumoperitoneum (3), pneumatosis intestinalis (5), thumbprinting (1), abscess and fistula (2), and intrahepatic gas (1). Pathological findings at operation or post-mortem examination showed ulcers, infarcts (the principal lesions in NEC), perforations, peritonitis, pneumatosis intestinalis, congested vessels, and inflammatory infiltrate in vessel walls. In all the present cases, the UV catheters had been introduced blindly and their position had not been localized radiologically. When the course of the catheter through the UV-ductus venosus (DV)-inferior vena cava (IVC) segment is followed radiologically, the properly positioned catheter viewed in the antero-posterior projection curves gently to the right of the mid-sagital line at T12 level before entering the DV. The lateral view determines the exact location of the catheter tip; when properly placed, it should be in the IVC or even the right atrium. The difficulty of properly positioning the UV catheter without radiological localization and the association of a malpositioned catheter with NEC are presented. The authors recommend radiological localization of a radiopaque catheter as a sensible prerequisite to beginning UV transfusion or infusion. They note that the radiographs can be taken with minimal handling of the patient on a portable x-ray machine.

8565 PERSISTENT POST-ENTERITIS DIARRHOEA. (Eng.) Zoppi, G.; Deganello, A.; Gaburro, D. (Cattedra di Auxologia and Clinica Pediatrica dell'Universita, Policlinico Borgo Roma, 37100 Verona, Italy). *Eur. J. Pediatr.* 126(4):225-236; 1977.

Two groups of pediatric patients--A, 18 patients <12 months old; B, 30 patients 12-30 months old--with chronic diarrhea following acute enteritis were evaluated clinically, biochemically, and gastroenterologically. The principal symptom in all patients was mild but persistent diarrhea for 4.5 ± 1.7 and 12.5 ± 4.9 months in groups A and B, resp. Growth was impaired in 12/18 patients in A and in 24/30 patients in B. Before hospitalization, all patients had been treated with antimicrobial drugs and had received milk-free, high-fiber diets. Laboratory studies gave the following results in groups A and B: mean hemoglobin, 10.4 ± 0.9 and 11.8 ± 1.1 g/dl, resp.; RBC, 4.1 4.1 ± 0.6 and 4.4 ± 0.2 10^6/ml, resp.; SGOT level, 75.7 ± 5.3 and 63.5 ± 15.6 IU/l, resp.; SGPT level, 37.7 ± 4.6 and 28.0 ± 4.3 IU/l, resp.; and iron level, 65.7 ± 26.5 and 62.4 ± 24.5 mg/dl, resp. Bone age retardation of -2.7 ± 1.9 and -5.3 ± 2.9 months in groups A and B, resp., was demonstrated. Stereomicroscopy of the intestinal mucosal biopsy specimens showed subtotal mucosal atrophy with ridges or convolutions, types II-c and II-c-d, in 9/18 patients in group A and in 10/30 patients in group B. Completely normal mucosa was never found. Liver involvement was excluded (normal SGPT values) and gut inflammation was confirmed (elevated SGOT values and study of biopsy material). Normal fecal flora were detected by bacteriological examination of the feces in all patients, but the total counts for both aerobic and anaerobic organisms in the patients were significantly ($p<0.01$) higher than normal, indicative of an altered ratio between the normal components of the fecal flora. A new diet was built up (polysaccharides, then proteins, disaccharides and dilute cow's milk); improvement began within 1 week. A normal diet, restricted only in high-fiber foods, was then adopted. Slow normalization of the stools occurred within 1-3 months, and weight and height approached normal at the end of 1 yr. After evaluation of the factors known to cause chronic inflammation after an acute infection, the authors conclude that the cause of persistent diarrhea in the patients was chronic inflammation due to the alteration of the intestinal microflora by inappropriate diet and therapy.

8566 THE GASTROINTESTINAL LESIONS IN LEUKEMIAS. (Jpn.) Hiroshige, Y.; Matsumoto, N.; Suetsugu, N.; Miwa, S.; Kaku, T.; Sasaki, K.; *et al.* (Yamaguchi Univ. Sch. Medicine, Japan). *Jpn. J. Clin. Hematol.* 18(7):861-867; 1977.

Gastrointestinal lesions were studied at autopsy in 51 cases of leukemia. The lesions included leukemic infiltration, ulcers, necrosis, and hemorrhage. Leukemic infiltration generally did not produce ulceration. There appeared to be a higher incidence of multiple ulcers (47.7%) in patients who had undergone combination chemotherapy. There was a frequent association between oral ulceration and bacterial or fungal infection at the site of the ulcers in these cases. These observations suggest that steroid therapy, combined chemotherapy, and gastrointestinal infection could be etiologic factors in multiple ulceration. Six patients had shown acute abdominal symptoms, consisting of oral ulceration, melena, shock, and marked neutropenia, at the terminal stage of their illness. Ileocecal or large intestinal necrosis associated with ulceration, pseudomembrane formation, and bacterial and/or fungus infection was observed in these patients. Gastrointestinal lesions were the direct cause of death in a total of 10 patients, with 8 dying from massive gastrointestinal hemorrhage.

8567 MORPHOLOGIC AND FUNCTIONAL STUDY OF THE SMALL INTESTINE IN MARASMIC PATIENTS. (Eng.) Fagundes Neto, U.; Wehba, J.; Patricio, F. R. S.; Machado, N. (Escola Paulista de Medicina, Rua Botucatu, 720, 04023, Sao Paulo, SP, Brazil). *Arq. Gastroenterol.* 14(4):241-248; 1977.

Jejunal morphology and small intestine function (absorbance of d-xylose, sucrose, lactose, and glucose) were studied in 22 children (3-36 months old) with marasmic malnutrition and chronic diarrhea (4 also had bronchopneumonia). Jejunal biopsy was performed after a minimum fast of 4 hr. The villus/crypt height ratio was used to classify the patients into four morphological classes (degree 0, I, II, or III) according to a published criterion; there were 2, 4, 14, and 2 patients in these categories, resp. An overall decrease in the number of villi was observed in 12 cases; in 6 cases, the epithelium had become cuboid and in 1 case, pseudostratified. The inflammatory infiltrate showed distinct degrees of alteration, but in all cases eosinophils were found on the lamina propria of the intestinal mucosa. The average values for d-xylose in the blood 1 and 2 hr after the patients had swallowed a d-xylose dose of 15 g/m^2 of body surface were 24.8 ± 9.2 and 28.0 ± 8.2 mg%; the dose was given after a minimum 4-hr fasting period. Also after a minimum 4-hr fast, overload tests for sucrose, lactose (2 g/kg body weight) and glucose (1 g/kg) were carried out on alternate days. Of 17 patients with normal sucrose and glucose absorption curves, 8 had normal lactose absorption curves and 9 had flat lactose absorption curves; 4 patients had normal glucose absorption curves but flat sucrose and lactose absorption curves; and the remaining patient had flat absorption curves for all three sugars. Three children died of complications (*Enterobacter* septicemia in 1 and bronchopneumonia in 2). This study has revealed morphologic functional alterations, termed "decreased villus population," similar to changes seen in kwashiorkor in children with marasmic malnutrition. The malabsorption of lactose in 73% of these patients emphasized the necessity of considering exclusion of cow's-milk formula during treatment. Intensive medical care and an adequate diet can reduce mortality and hospitalization time in these patients.

8568 THE ZOLLINGER-ELLISON SYNDROME. REPORT OF ONE CASE AND REVIEW OF THE LITERATURE. (Fre.) Sarrazin, R.; Dyon, J. F.; Bost, M.; Rossignol, A. M.; Rachail, M.; Fournet, J.; *et al.* (Centre Hospitalier Universitaire de Grenoble, 38700 La Trouce, France). *Ann. Pediatr.* 53(12): 863-874; 1977.

A case is reported of a 9-yr-old girl with the Zollinger-Ellison (Z-E) syndrome that was revealed by a bleeding duodenal ulcer, and reports in the literature on 19 children under 15 yr of age with the Z-E syndrome are reviewed. The patient presented with episodic abdominal pain unrelated to meals. Initial x-ray studies and a laparotomy failed to disclose any abdominal pathology. An ulcer of the duodenum was revealed on barium transit studies taken after the patient had two episodes of vomiting with hematemesis. The Z-E syndrome was identified by radioimmunoassay studies of plasma gastrin performed after fasting, ingesting a high protein meal, or receiving i.v. calcium (12 mg/kg). A nontumorous hepatomegaly and the duodenal ulcer were observed during a second laparotomy. At first, the pancreas appeared normal, but careful exploration disclosed an enlarged lymph node on the posterior surface of the pancreas next to the duodenum. A total gastrectomy and excision of the head of the pancreas and adjoining duodenum were performed. Histological examination of the excised pancreas did not reveal any abnormal parenchyma; however, the retropancreatic lymph nodes were invaded by an islet cell tumor. Serum gastrin levels fell to normal postoperatively, and 27 months later remained low. The literature reports only 7/19 children with Z-E syndrome with tumors located in the pancreas; other sites included lymph nodes (6), liver (8), and the gastrointestinal tract (2).

8569 IRON METABOLISM DURING PROTEIN-ENERGY MALNUTRITION IN CHILDREN. (Fre.) Fondu, P.; Bouton, J. M.; de Maertelaere-Laurent, E.; Mandelbaum, I. M. (Hopital Saint-Pierre, Rue Haute, 320, B 1000 Bruxelles, Belgium). *Nouv. Rev. Fr. Hematol. Blood Cells* 18(1):5-21; 1977.

8570 QUANTITATIVE INVESTIGATIONS ON THE ENTERAL RESORPTION OF INHALED ALLERGENS. (Ger.) Jorde, W.; Linsenmann, P.; Werdermann, K.; Bohlmann, H. G. (Asthmakrankenhaus der Kamillianer, Monchengladbach, W. Germany). *Z. Immunitaetsforsch.* (Suppl. 2):135-138; 1977.

8571 EXUDATIVE GASTROENTEROPATHY REVEALING ALLERGY TO CRUSTACEANS. (Fre.) Dulac, O.; Zamet, P.; Bonnefond, A. (Hopital d'enfants de Bicetre, F 94270 Bicetre, France). *Arch. Fr. Pediatr.* 34(5):455-456; 1977.

8572 ENDOSCOPIC INJECTION OF SCLEROSING AGENTS FOR DIGESTIVE TRACT HEMOSTASIS. (Ger.) Soehendra, N.; Knipper, A. (Chirurgische Klinik und Poliklinik der Universitat, Martinistrasse 52, 2000 Hamburg 20, W. Germany). *Dtsch. Med. Wochenschr.* 102(46):1688-1690; 1977.

8573 TACTICAL ERRORS IN SURGICAL MANAGEMENT OF ACUTE UPPER GASTROINTESTINAL HEMORRHAGE. (Ger.) Schnetzer, J. (Krankenhaus der Barmherzigen Schwestern, Liniengasse 19, A-1062 Vienna, Austria). *Wien. Med. Wochenschr.* 127(24):744-745; 1977.

8574 DRUG-INDUCED HEMORRHAGE OF THE UPPER PART OF THE DIGESTIVE TRACT. (Fre.) Doffoel, M.; Coumaros, D.; Girard, M.; Krummel, Y.; Bockel, R. (Centre Hospitalo-Universitaire de Strasbourg, 67005 Strasbourg Cedex, France). *Rev. Fr. Gastroenterol.* (134):23-28; 1977.

8575 INTESTINAL DYSBACTERIOSIS IN CHILDREN. (Rus.) Dorofeichuk, V. G. (Gorkii Scientific Res. Inst. Pediatrics, RSFSR Ministry Public Health, Gorkii, USSR). *Vopr. Okhr. Materin. Det.* 22(2):58-60; 1977.

8576 EFFECTIVENESS OF INTESTOPANE AND OTHER ETIOTROPIC DRUGS IN ACUTE DYSENTERY. (Rus.) Chaitsev, V. G.; Chechel'nitskii, V. M.; Terent'ev, V. F. (Voronezh Medical Inst., Voronezh, USSR). *Sov. Med.* (11):149-151; 1977.

8577 SIGNIFICANCE OF DETERMINATION OF DNA IN THE JEJUNUM CONTENTS FOR DIAGNOSIS OF CHRONIC ENTERITIS. (Rus.) Linevsky, Yu. V. (Dept. Therapy, A. M. Gorkii Donetsk Medical Inst., Donetsk, USSR). *Ter. Arkh.* 49(2):52-55; 1977.

8578 X-RAY DIAGNOSIS OF CHRONIC NONSPECIFIC ENTEROCOLITIS. (Ger.) Frik, W.; Persigehl, M. (Medizinische Fakultat, Rheinisch-Westfalische Technische Hochschule, Goethestrasse 27/29, 5100 Aachen, W. Germany). *Therapiewoche* 27(38):6683-6690, *passim*; 1977.

8579 CONSTIPATION IN CHILDREN. ITS TREATMENT WITH BRAN. (Fre.) Bargheon, J. (No affiliation given). *Rev. Fr. Gastroenterol.* (132):5-12; 1977.

8580 TREATMENT OF CONSTIPATION WITH BRAN CAKES. (Fre.) Choffe, R. (Clinique Saint-Dominique, 71500 Louhans, France). *Rev. Fr. Gastroenterol.* (134):5-7; 1977.

8581 THERAPY FOR OBSTIPATION. (Ger.) Berg, H. (2357 Bad Bramstedt, W. Germany). *Therapiewoche* 27(29):5353-5358; 1977.

8582 THERAPEUTIC TRIAL WITH NITROXOLINE-CHARCOAL IN CERTAIN DIARRHEAS. (Fre.) Paris, J. (No affiliation given). *Rev. Fr. Gastroenterol.* (131):65-71; 1977.

8583 DIPHENOXINE HYDROCHLORIDE, A NEW ANTIDIARRHEAL AGENT. (Ger.) Steffen, R. (Spital Neumunster, Zollikerberg bei Zurich, Switzerland). *Munch. Med. Wochenschr.* 120(2/3):73-74; 1978.

8584 SALMONELLOSIS. (Ger.) Werner, G. T. (Bayerischen Landesimpfanstalt, Tropenmed. Beratungsstelle, Am Neudeck 1, 8 Munich 95, W. Germany). *Fortschr. Med.* 95(36):2189-2194; 1977.

8585 EPIDEMIOLOGICAL ASPECTS OF SALMONELLOSIS. (Ger.) Hupper, H. (Ministerium fur Arbeit, Gesundheit und Sozialordnung, Rotebuhlplatz 30, 7000 Stuttgart, W. Germany). *Therapiewoche* 27(29):5298-5303, *passim*; 1977.

8586 NOTES ON 560 CASES OF SALMONELLOSIS. (Ita.) Brunet, M. R.; Giachino, M.; Tapparo, D.; Marcer, I.; Vaccino, P. (Sch. Puericulture, Univ. Turin, Turin, Italy). *Minerva Pediatr.* 29(1):9-18; 1977.

8587 WIDESPREAD SALMONELLOSIS IN A PEDIATRIC WARD. OBSERVATION OF 261 CASES. (Fre.) Estavoyer, J. M.; Litzler, B.; Dupont, M. J.; Bruand, L.; Baufle, G. H.; Raffi, A. (Centre Hospitalier et Universitaire, 25000 Besancon, France). *Rev. Fr. Gastroenterol.* (130):15-28, *passim*; 1977.

8588 EPIDEMIOLOGICAL AND CLINICAL FEATURES OF Salmonella-INDUCED ENTERITIS. (Ita.) Rossolini, A.; Mattei, C.; Bianchini, A. M. (Istituto di Clinica delle Malattie Infettive dell'Universita di Siena, Siena, Italy). *Minerva Pediatr.* 29(1):1-7; 1977.

8589 PROBLEM OF CHRONIC CARRIERS IN Salmonella INFECTION. (Ger.) Petersen, K. F. (Zentralkrankenhaus Gauting, Unterbrunner Strasse 85, 8035 Gauting, W. Germany). *Therapiewoche* 27(29):5304-5307, *passim*; 1977.

8590 CLINICAL-IMMUNOLOGICAL CHARACTERISTICS OF GASTROINTESTINAL SALMONELLOSIS IN CHILDREN OF DIFFERENT AGE GROUPS. (Rus.) Rumiantseva, T. A. (Dept. Infectious Diseases of Children, Central Inst. Postgraduate Medical Studies, USSR). *Pediatriia* (7):63-68; 1977.

8591 EFFECT OF AZAPROPAZONE ON THE GASTROINTESTINAL TRACT AND LIVER. (Ger.) Faust-Tinnefeldt, G. (Zentrum fur Rheumatologie, Medizinische Klinik, 6229 Schlangenbad, W. Germany). *Therapiewoche* 27(47):8567-8575, *passim*; 1977.

8592 METASTASES OF CYLINDROME OF THE SUBLINGUA GLAND TO THE SCALP. (Ger.) Reichelt, H. G.; Savaser, A. (Medizinische Hochschule Hannover, D-3000 Hannover 61, W. Germany). *Fortschr. Geb. Rontgenstr. Nuklearmed.* 128(1):96-97; 1978.

8593 GASTROENTERITIS IN ALICE SPRINGS. (Eng.) Gardiner, A. J. (Adelaide Children's Hosp 72 King William Rd., North Adelaide, South Australi 5006, Australia). *Med. J. Aust.* 4(2, Suppl.):6-10; 1977.

8594 ONCOCYTOMA OF THE PAROTID GLAND WITH MALIGNANT CHANGE. (Eng.) Chu, W.; Strawitz, J. G. (American Oncologic Hosp., Central and Shelmire Ave., Philadelphia, PA 19111). *Arch. Surg.* 113(3):318-319; 1978.

8595 THE TOXICITY OF ETHANOL. A TENTATIVE RISK EVALUATION. (Eng.) Rydberg, U.; Skerfving, S. (Karolinska Institutet, S-104 01 Stockholm, Sweden). *Adv. Exp. Med. Biol.* 85b:403-419; 1977.

8596 ERRORS IN REPORTING CANCER AND OTHER CONDITIONS BY PERSONS IN A PROSPECTIVE STUDY. (Eng.) Aono, J.; Nomura, A. (Kuakini Medical Center, Honolulu, HI). *Publ. Health Rep.* 93(1):11-15; 1978.

8597 ENTEROVIRAL DISEASE IN EARLY INFANCY. (Eng.) Morens, D. M. (Viral Diseases Div., Building 1, Room 6124, Bureau Epidemiology, Center Disease Control, Atlanta, GA 30333). *J. Pediatr.* 92(3):374-377; 1978.

See also, 8017, 8137, 8140, 8142, 8153, 8155, 8163, 8167, 8172, 8173, 8180, 8194, 8212, 8217, 8228, 8248, 8298, 8302, 8318, 8328, 8355, 8365, 8372, 8384, 8396, 8410, 8432.

GENERAL

Parasitology

8598 THE ENZYME-LINKED IMMUNOSORBENT ASSAY (ELISA) IN THE DIAGNOSIS OF AMOEBIASIS. (Ita.) Sorice, F.; Delia, S.; Vullo, V.; Ferone, U. (Istituto di Clinica delle Malattie Tropicali e Infettive, Universita di Roma, Roma, Italy). *Ann. Solavo* 19(3):484-489; 1977.

The application of the new serological method, the enzyme-linked immunosorbent assay (ELISA), in the detection of *Entamoeba histolytica* antibodies is reported for 39 subjects (15 harboring *E. histolytica* trophozoites, 6 with acute enteric symptomatology of undiagnosed cause, 8 with other parasitic infections, and 10 healthy individuals). The test is performed by incubating dilute antigen with the serum to be tested for 3 hr at 37 C. A total human antiglobulin antiserum conjugated with alkaline phosphatase is added followed by the enzyme and, in approximately 15-30 min, positive sera are distinguished by their intense yellow color. The results are evaluated with a spectrophotometer. Of sera from 15 patients with *E. histolytica* trophozoites, the optical density (O.D.) values were 0.42-0.80 in 9 cases and 0.81-0.90 in 6 cases (the average value for this group was 0.70). In six patients with enteric symptoms, the O.D. values were 0.15-0.20 with a median value of 0.17. The eight patients with other parasitic infections had O.D. values of 0.12-0.20 with an average value of 0.18. The average O.D. value of the control group was 0.12. The differences in the values between those with *E. histolytica* infections, those with other infections, and the control group were statistically significant ($p<0.001$). It is concluded that the ELISA is useful in the diagnosis of *E. histolytica* infections.

8599 COMPARATIVE EVALUATION OF TINIDAZOLE AND METRONIDAZOLE IN THE TREATMENT OF AMOEBIC LIVER ABSCESS. (Eng.) Kundu, S. C.; Sen, A.; Bhattacherjee, T. D.; Dasgupta, D. P.; Majumdar, S.; Mookerjee, P. K.; *et al.* (Singh Hosp., E. Rly., Calcutta, India). *J. Indian Med. Assoc.* 69(6): 127-129; 1977.

A randomized comparative clinical study of tinidazole (T) and metronidazole (M) in comparable cases of amebic liver abscess was carried out to evaluate objectively the therapeutic effect of, tolerance to, and acceptibility of each drug. Patients with clinical features of liver abscess but without serious complications and not moribund were selected for the study; further, these subjects either had not received or had failed to respond to any anti-amebic drug during the previous month. Nine patients each received either T or M (2 g once daily for 3 days, p.o., for both drugs), chloroquine, and/or emetine HCl, if necessary, to obtain a cure, and supportive nonspecific treatment as needed. Clinical assessment was carried out prior to and on the 5th, 10th, and 30th days after treatment; therapeutic response was graded excellent, good, fair, or poor according to rapidity of response to the test drug, time until clinical cure, and need for auxiliary specific and/or nonspecific treatment. The responses to T were significantly different from those to M (excellent, 7 and 2; good, 1 and 1; fair, 0 and 0; poor, 1 and 6; resp., $p<0.05$ for excellent and good versus fair and poor). One patient in each test group died. One patient in the T group and four in the M group complained of side-effects (anorexia in both groups; nausea, giddiness, headache, and sleeplessness in the M group). Tinidazole is superior to metronidazole, and therefore is the drug of choice, in treating uncomplicated amebic liver abscess.

8600 BILIARY ASCARIASIS. (Eng.) Arcilla, C. A.; Varilla, A. (Ramon Magsaysay Memorial Medical Center, Quezon City, Philippines). *Drugs* 15(Suppl. 1):104-110; 1978.

8601 TINIDAZOLE IN THE TREATMENT OF TRICHOMONIASIS, GIARDIASIS AND AMOEBIASIS: REPORT OF A MULTICENTRE STUDY. (Eng.) Apte, V. V.; Packard. R. S. (Medical Dept., Pfizer Asia, G.P.O. Box 573, Hong Kong). *Drugs* 15(Suppl. 1): 43-48; 1978.

8602 HOW DOES TINIDAZOLE COMPARE WITH METRONIDAZOLE? A SUMMARY REPORT OF INDIAN TRIALS IN AMOEBIASIS AND GIARDIASIS. (Eng.) Bakshi, J. S.; Ghiara, J. M.; Nanivadekar, A. S. (Pfizer Ltd., Express Towers, Nariman Point, Bombay 400 021, India). *Drugs* 15(Suppl. 1):33-42; 1978.

8603 SINGLE DOSE THERAPY OF GIARDIASIS WITH TINIDAZOLE AND METRONIDAZOLE. (Eng.) Gazder, A. J.; Banerjee, M. (Calcutta Natl. Medical Coll., Calcutta, India). *Drugs* 15(Suppl. 1):30-32; 1978.

8604 TINIDAZOLE AND METRONIDAZOLE IN HEPATIC AMOEBIASIS. (Eng.) Islam, N.; Hasan, K. (Inst. Postgraduate Medicine & Res., Dacca, Bangladesh). *Drugs* 15(Suppl. 1):26-29; 1978.

8605 TREATMENT OF AMOEBIC LIVER ABSCESS WITH TINIDAZOLE AND METRONIDAZOLE. (Eng.) Khokhani, R. C.; Garud, A. D.; Deodhar, K. P.; Sureka, S. B.; Kulkarni, M.; Damle, V. B. (Lokmanya Tilak Municipal Medical Coll. and Hosp., Bombay, India). *Drugs* 15(Suppl. 1):23-25; 1978.

8606 A COMPARATIVE STUDY OF TINIDAZOLE AND METRONIDAZOLE AS A SINGLE DAILY DOSE FOR THREE DAYS IN SYMPTOMATIC INTESTINAL AMOEBIASIS. (Eng.) Misra, N. P. (Gandhi Medical Coll., Bhopal 462 002, India). *Drugs* 15(Suppl. 1):19-22; 1978.

See also, 7999, 8100, 8139, 8267, 8339, 8371, 8593.

GENERAL

Regional Enteritis

8607 CROHN'S DISEASE: A LONG-TERM STUDY OF THE CLINICAL COURSE IN 186 PATIENTS. (Eng.) Bergman, L.; Krause, U. (Univ. Hosp., Uppsala, Sweden). *Scand. J. Gastroenterol.* 12(8): 937-944; 1977.

A series of 186 patients with Crohn's disease treated during the period 1956-1968 was followed up. During the follow-up period, 177 patients had undergone resection or bypass surgery. The mean observation time in these patients was 10.5 yr. The overall recurrence rate after resection was 51%. Radical resection (i.e., all affected areas resected together with a margin of at least 10 cm of microscopically normal tissue) was carried out in 84 patients, and the recurrence rate was 29%. Among the 57 patients who underwent nonradical resection (i.e., all diseased bowel was not removed or else a disease-free margin of less than 10 cm on either side of the resection remained), the recurrence rate was 84%. Patients who had undergone a primary radical resection needed fewer re-operations and had fewer recurrences and a better quality of life than patients with a nonradical first operation. The total mortality in operations or due to complications of Crohn's disease was 5.4%. The mortality in connection with the operative procedure was 3.4%. The general health of the 165 patients examined in 1975 was estimated: 87% were in very good general health, 9.1% had moderate subjective symptoms, and 3.6% had pronounced subjective symptoms.

8608 REGIONAL ENTERITIS IN CHILDREN: CLINICAL AND ROENTGEN FEATURES. (Eng.) Franken, E. A., Jr.; Smith, J. A.; Fitzgerald, J. R. (Indiana Univ. Sch. Medicine, Indianapolis, IN). *CRC Crit. Rev. Diagn. Imaging* 10(2):163-185; 1977.

A study of regional enteritis (RE) or Crohn's disease diagnosed in 36 children at Riley Children's Hospital of Indiana University is presented. The clinical manifestations of the disease, type of involvement, gastrointestinal and systemic complications, roentgenology, and problems of differential diagnosis in these children and in 268 pediatric RE cases in the literature are compared and frequently contrasted with related findings in over 600 adult RE cases in the literature. About one-third of the 36 children initially presented systemic problems such as arthritis (3), growth failure (2), fever of unknown origin (4), anorexia nervosa (2), and other nongastrointestinal problems (3). Nonspecific abdominal pain occurred in over 80% of the cases. The disease was limited to the small bowel in 13 of the children and in 4 of these there was no involvement of the terminal

ileum. Nine of the 36 had isolated colon involvement. The authors emphasize that in a child with gastrointestinal symptoms requiring roentgenographic investigation, it is of greater importance to examine the small bowel and colon for possible RE than to examine for duodenal and gastric ulcer. Where RE is suspected, a complete roentgenographic evaluation including standard barium studies of the stomach, small bowel, and colon are indicated initially with retrograde small bowel examination to confirm the diagnosis in borderline cases and to assess the extent of involvement. Once the diagnosis of RE is established, techniques of examination can be modified to reduce radiation exposure.

8609 ISOLATED COLONIC CROHN'S DISEASE: CLINICAL, RADIOLOGICAL, AND ENDOSCOPIC ASPECTS, DIAGNOSIS, AND EVOLUTION. (Fre.) Julien, M.; Vignal, J. (Hopital Henri-Mondor, Creteil, France). *Rev. Prat.* 27(55):3817-3834, *passim*; 1977.

Various aspects of Crohn's disease (CD) are reviewed. Colonic CD can be segmental, multisegmental, or diffuse. The rectum is involved together with other areas of the colon in 35-40% of cases. Rectal involvement alone is exceptional and occurs in only 2-3% of cases. Diarrhea accompanied by abdominal cramps, and sometimes containing pus, are the usual clinical signs. Rectal bleeding is much more likely to occur with rectal involvement. Differential diagnosis of CD and other colitis, particularly ulcerative colitis, is discussed. Barium transit x-ray studies identify the type of lesions (ulcerations, "thumb-print" lacunae) and site of lesions (right colon, ileum). X-ray often fails to identify rectal lesions, which can be found by endoscopic examination and biopsy. The evolution of the disease is highly variable and difficult to predict. Complications such as occlusions, fistulae, abscesses, and colectasia occur frequently, and surgery may be required; however, pathology will recur in 50% of patients.

8610 DETERMINATION OF THE FAECAL EXCRETION OF LABELLED BILE SALTS AFTER I.V. ADMINISTRATION OF ^{14}C-CHOLIC ACID: AN EVALUATION OF THE BILE SALT MALABSORPTION BEFORE AND AFTER SURGERY IN PATIENTS WITH CROHN'S DISEASE. (Eng.) Andersson, H.; Filipsson, S.; Hulten, L. (Sahlgrenska sjukhuset, S-413 45 Goteborg, Sweden). *Scand. J. Gastroenterol.* 13(2):249-255; 1978.

The value of a recently described technique for determining fecal bile salt loss in the study of ileal function was assessed. By measuring total fecal radioactivity, correlated to 24-hr enterohepatic circulation, following the i.v. administration of ^{14}C-cholic acid, bile salt malabsorption was evaluated before and/or after surgery in 80 patients with ileal or colonic Crohn's disease, and the results were related to the length of ileum diseased or resected. Before surgery, bile salt malabsorption was observed only in nine patients with terminal ileal inflammation, but no significant correlation was found between bile salt excretion and the extent of ileal disease. Twenty-one patients with ileal, 36 patients with ileocolic, and 9 patients with colonic disease were investigated after surgery. The percentage of administered bile salt excreted in the feces correlated well with the length of ileum resected ($r=0.79$, $p<0.001$). The correlation between fecal bile salt level and the length of the resected segment expressed in percentage of total small bowel length was of the same magnitude ($r=0.80$, $p<0.001$). The correlation between bile salt malabsorption and the length of ileal segment resected was about the same in ileostomy patients and in patients subjected to restorative operation. In 19 patients investigated both pre- and postoperatively, it was possible to compare directly the magnitude of the bile salt loss caused by the ileal disease and that caused by the resection. Irrespective of the preoperative level, the small bowel resection resulted in an increased loss of bile salts ($p<0.001$). When a similar comparison was made for fecal fat excretion and the results of the Schilling test, the results were less consistent. Three ileal function tests were carried out in 50 patients with malabsorption due to resection of less than 10 cm of ileum. The Schilling test gave abnormal results in only 48%, whereas a fecal fat excretion test confirmed malabsorption in 64%, and the fecal bile salt test gave results indicative of malabsorption in 96%. The agreement between the three tests improved in patients with ileal resections of more than 60 cm. It is concluded that the determination of fecal ^{14}C-cholic acid is a more sensitive test than the Schilling test or the fecal fat excretion test, at least in those patients with ileal dysfunction due to ileal resection.

8611 TREATMENT OF CROHN'S DISEASE IN THE CHILD. 12 CASES. (Fre.) Navarro, J.; Fontaine, J. L.; Mathe, J. C.; Girardet, J. P.; Pernin, P.; Mougenot, J. F.; *et al.* (Hopital Trousseau, 24 av. du Dr. A. Netter, F 75012, Paris, France). *Nouv. Presse Med.* 7(3):183-188; 1978.

The results of the treatment of Crohn's disease in 12 children (7 boys, 5 girls; age range 6-17 yr) are reported. In the five patients treated prior to 1974, treatment consisted of a low-residue diet, milk deprivation, and sometimes i.v. feeding. Salazosulfapyridine (100 mg/kg/day) and corticosteroid therapy (1.5-3 mg/kg/day) for 15 days/month were prescribed for four of five patients. Three of the five patients eventually required surgery because of stenosis (2) or fistula (1). The more recently treated group of seven patients were fed by i.v. and/or constant-rate enteral feeding regimens for periods of 15-22 days and 15-510 days (average 120 days), resp. Constant-rate nutrition by gastric intubation was possible with the patient at home in three cases by use of portable pump. A low-residue, hypoallergenic diet was instituted at the end of parenteral and/or enteral feeding. Salazosulfamide (100 mg/kg/day), metronidazole (20 mg/kg), and levamisole (3 cases only, dose not specified) were prescribed for these patients. Five of the seven patients in the recently treated group, followed

for more than 1 yr, demonstrated a rapid weight gain, disappearance of diarrhea and cramps, healing of mucosal fissures and anorectal fistulas, and in one case, disappearance of duodenal stenosis. Enteral feeding was not well tolerated by adolescents in the group; however, children were more likely to accept medications and protein hydrolysates by this route.

8612 RESULTS OF TREATING THE ACUTE STAGE OF CROHN'S DISEASE BY A DIETARY REGIMEN. (Ger.) Fromm, H.; Gebel, M.; Schroeter, U.; Canzler, H.; Schmidt, W. (Medizinische Hochschule, Karl-Wiechert-Allee 9, 3000 Hannover 61, W. Germany). *Dtsch. Med. Wochenschr.* 103(9):377-380; 1978.

The effectiveness of diet treatment during the acute phase of Crohn's disease (CD) was studied in a series of 23 patients. Fifteen patients received at first exclusively parenteral feeding (2,500 kcal/day; 20% amino acids, 25% fats, 55% carbohydrates); the others received a balanced synthetic diet (Vivasorb) through a nasoduodenal probe. A low-residue diet was then introduced gradually in all patients. The cardinal symptoms of CD such as severe abdominal pain, diarrhea, incomplete ileus, and weight loss, responded favorably in all patients. Postoperative fistulae closed in two of three patients, but enterocutaneous fistulae remained open in all of five patients, although the volume of secretion decreased distinctly in four. During the follow-up (1-24 months, average 9 months), the symptoms recurred in five patients, necessitating operative treatment in three. The results indicate that diet treatment of CD is an effective therapy.

8613 THE DILEMMA OF CROHN'S DISEASE: LONG-TERM FOLLOW-UP OF CROHN'S DISEASE OF THE SMALL INTESTINE. (Eng.) Stone, W.; Veidenheimer, M. C.; Corman, M. L.; Coller, J. A. (Lahey Clinic Foundation, 605 Commonwealth Ave., Boston, MA 02215). *Dis. Colon Rectum* 20(5):372-376; 1977.

8614 CROHN'S DISEASE OF THE DUODENUM [Abstract]. (Eng.) Anonymous. (No affiliation given). *Br. Med. J.* 2(6082):282; 1977.

8615 RECTAL BIOPSY IN CROHN'S DISEASE [Abstract]. (Eng.) Hill, R. B.; Kent, T. H.; Hansen, R. (Upstate Medical Center, IA). *Gastroenterology* 72(5, Part 2):1069; 1977.

8616 ABNORMAL JEJUNAL SURFACE pH IN CROHN'S DISEASE--NEW EVIDENCE THAT CROHN'S DISEASE IS A DIFFUSE LESION OF THE GASTROINTESTINAL TRACT [Abstract]. (Eng.) Cooper, B. T.; Lucas, M. L.; Lei, F. H.; Blair, J. A.; Cooke, W. T. (General Hosp., Birmingham, England). *Gut* 18(5):A423; 1977.

8617 BONE HISTOLOGY AND VITAMIN D STATUS IN CROHN'S DISEASE (CD): ASSESSMENT OF VITAMIN D THERAPY [Abstract]. (Eng.) Driscoll, R.; Meredith, S.; Wagonfeld, J.; Rosenberg, I. (Dept. Medicine, Univ. Chicago, Chicago, IL). *Gastroenterology* 72(5, Part 2):1051; 1977.

8618 THYMOSIN EFFECT ON 'ACTIVE' T-LYMPHOCYTES IN CROHN'S DISEASE [Abstract]. (Eng.) Dopp, A. C.; Goldstein, A. L.; Mutchnick, M. G. (Dept. Internal Medicine, Univ. Michigan, Ann Arbor, MI). *Gastroenterology* 72(5, Part 2):1051; 1977.

8619 RENAL TUBULAR ACIDOSIS SUPERIMPOSED ON THE "SHORT BOWEL" SYNDROME IN CROHN'S DISEASE [Abstract]. (Eng.) Singh, P.; Katz, S.; Katzka, I. (Long Island Jewish-Hillside Medical Center, New Hyde Park, NY). *Gastroenterology* 72(5, Part 2): 1132; 1977.

See also, 8143, 8304, 8348, 8349, 8388, 8391, 8395.

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